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THE EFFECTS OF EXERCISE UPON
SKELETAL MUSCLE GLYCOGEN SYNTHETASE ACTIVITY

by

© ROBERT EDWARD THAYER

A THESIS

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled "The Effects of Exercise Upon Skeletal Muscle Glycogen Synthetase Activity", submitted by Robert Edward Thayer in partial fulfilment of the requirements for the degree of Master of Science.

ABSTRACT

The glycogen synthetase activity of working skeletal muscle and the adaptations which occurred as a result of exercise and training were examined. A group of eighteen male subjects from the University of Alberta were divided into sedentary and active groups. Prior to the actual experiment all subjects were tested for MVO_2 on a bicycle ergometer. A work load of 70% MVO_2 was then calculated for the submaximal test. The work load for the maximal test was set at 100% MVO_2. All subjects underwent both the submaximal and maximal tests during which, muscle biopsies were taken from the vastus lateralis. In the submaximal test, biopsies were taken at rest, after every twenty minutes of work, at the point of fatigue and after a ten minute recovery period. For the maximal test biopsies were taken at rest, at the point of fatigue and following a ten minute recovery. Simultaneously as muscle samples were taken, 5.0 ml samples of blood were withdrawn from the brachial vein. The active group, which was comprised of wrestlers and track men, trained for four to five months in their respective sports and then were retested on all physiological measures taken prior to the training regimen. At the cessation of all testing, muscle samples were analyzed for glycogen synthetase activity and glycogen content. Blood samples were analyzed for glucose and lactic acid.

For the submaximal work loads a significant decrease in glycogen synthetase D activity for the post-training group was observed. No significant differences were noted for fatigue versus recovery. Initial glycogen synthetase D activity of the post-training group was significantly greater than initial values of the sedentary group. Glycogen synthetase activity of the post-training group was in most cases higher than initial values of the sedentary or pre-training groups. For the maximal test no significant decreases in glycogen synthetase I and D activity from initial to fatigue were observed. As in the submaximal test, the post-training group exhibited the highest initial glycogen synthetase I and D values. From fatigue to recovery there was a significant increase in synthetase I activity for the post-training group. Muscle glycogen synthetase I and D values at the point of fatigue were lower for the submaximal than for the maximal test.

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LIST OF FIGURES

FIGURE PAGE

1. Glucose and α1,4 and α1,6 Linkage . . . 5
2. Chart for the Glycolytic Pathway 6
3. Chart for the Krebs Cycle 9
4. Chart for Glycogen Synthesis and Breakdown 11
5. Glycogen Synthetase D Activity of all Groups for Submaximal Work Loads . . . 35
6. Glycogen Synthetase D Activity of all Groups for Maximal Work Loads 36
7. Glycogen Synthetase I Activity of all Groups for Submaximal Work Loads . . . 37
8. Glycogen Synthetase I Activity of all Groups for Maximal Work Loads 38
9. Data Sheet 65
10. Time Course for Synthetase D Solution . . 66

LIST OF TABLES

TABLE PAGE

1. Analysis of Variance for Glycogen Synthetase I: Submaximal: Pre vs. Post 40

2. Analysis of Variance for Glycogen Synthetase I: Maximal: Pre vs. Post 40

3. Analysis of Variance for Glycogen Synthetase D: Submaximal: Pre vs. Post 41

4. Analysis of Variance for Glycogen Synthetase D: Maximal: Pre vs. Post 41

5. Analysis of Variance for Glycogen Synthetase I: Submaximal: Sed vs. Post 42

6. Analysis of Variance for Glycogen Synthetase I: Maximal: Sed vs. Post 42

7. Analysis of Variance for Glycogen Synthetase D: Submaximal: Sed vs. Post 43

8. Analysis of Variance for Glycogen Synthetase D: Maximal: Sed vs. Post 43

9. Q Values: Glycogen Synthetase D: Submaximal: Sed vs. Post 45

10. Q Values: Glycogen Synthetase D: Maximal: Sed vs. Post 45

11. Q Values: Glycogen Synthetase I: Submaximal: Sed vs. Post 46

12. Q Values: Glycogen Synthetase I: Maximal: Sed vs. Post 46

13. Q Values: Glycogen Synthetase D: Submaximal: Pre vs. Post 47

14. Q Values: Glycogen Synthetase D: Maximal: Pre vs. Post 47

15. Q Values: Glycogen Synthetase I: Submaximal: Pre vs. Post 48

TABLE PAGE

16. Q Values: Glycogen Synthetase I: Maximal: Pre vs. Post 48

17. Tests on Overall Differences: Pre vs. Post . 50

18. Tests on Overall Differences: Sed vs. Post . 50

19. Table of Age, Weight, Height, Submaximal and Maximal Work Loads, MVO_2 and Submaximal and Maximal Work Times 67

20. Consent Form 70

21. Muscle Glycogen Synthetase D Activity for the Submaximal Test 71

22. Muscle Glycogen Synthetase I Activity for the Submaximal Test 73

23. Blood Glucose in MG. % for the Submaximal Test 74

24. Blood Lactate in MG. % for the Submaximal Test 76

25. Muscle Glycogen Synthetase D Activity for the Maximal Test 78

26. Muscle Glycogen Synthetase I Activity for the Maximal Test 79

27. Blood Glucose in MG% for the Maximal Test . . 80

28. Blood Lactate in MG% for the Maximal Test . . 81

29. Summary of Analysis of Variance: Sed vs. Post: Submaximal Glycogen Synthetase D . . 83

30. Summary of Analysis of Variance: Sed vs. Post: Maximal Glycogen Synthetase D 83

31. Summary of Analysis of Variance: Sed vs. Post: Submaximal Glycogen Synthetase I . . 84

32. Summary of Analysis of Variance: Sed vs. Post: Maximal Glycogen Synthetase I . . . 84

33. Summary of Analysis of Variance: Pre vs. Post: Submaximal Glycogen Synthetase D . . 85

TABLE PAGE

34. Summary of Analysis of Variance: Pre vs. Post: Maximal Glycogen Synthetase D . . . 85

35. Summary of Analysis of Variance: Pre vs. Post: Submaximal Glycogen Synthetase I . . 86

36. Summary of Analysis of Variance: Pre vs. Post: Maximal Glycogen Synthetase I . . . 86

37. Multiple Comparisons: Glycogen Synthetase D Submaximal: Sed vs. Post 87

38. Multiple Comparisons: Glycogen Synthetase D Maximal: Sed vs. Post 88

39. Multiple Comparisons: Glycogen Synthetase I Submaximal: Sed vs. Post 89

40. Multiple Comparisons: Glycogen Synthetase I Maximal: Sed vs. Post 90

41. Multiple Comparisons: Glycogen Synthetase D Submaximal: Pre vs. Post 91

42. Multiple Comparisons: Glycogen Synthetase D Maximal: Pre vs. Post 92

43. Multiple Comparisons: Glycogen Synthetase I Submaximal: Pre vs. Post 93

44. Multiple Comparisons: Glycogen Synthetase I Maximal: Pre vs. Post 94

45. Test on Overall Differences: Sed vs. Post Submaximal: Glycogen Synthetase D 95

46. Test on Overall Differences: Sed vs. Post Maximal: Glycogen Synthetase D 95

47. Test on Overall Differences: Sed vs. Post Submaximal: Glycogen Synthetase I 95

48. Test on Overall Differences: Sed vs. Post Maximal: Glycogen Synthetase I 96

49. Test on Overall Differences: Pre vs. Post Submaximal: Glycogen Synthetase D 96

TABLE PAGE

50. Test on Overall Differences: Pre vs. Post Maximal: Glycogen Synthetase D 96

51. Test on Overall Differences: Pre vs. Post Submaximal: Glycogen Synthetase I 97

52. Test on Overall Differences: Pre vs. Post Maximal: Glycogen Synthetase I 97

CHAPTER I

INTRODUCTION

Glycogen, a highly branched polysaccharide, is the principle storage form of carbohydrate in man and animals. Within the body, glycogen is found mainly in the skeletal muscle and liver. During exercise, when energy is required, the glycogen in muscle is broken down by the actions of the debranching enzyme and phosphorylase-a into *G-1-P. The G-1-P is then converted to G-6-P which follows the many reactions of the glycolytic pathway to pyruvate or lactic acid. If there is a sufficient supply of oxygen, pyruvate is not converted to lactate but enters the Citric Acid Cycle for a maximum yield of energy in the form of ATP. The ATP

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*The abbreviations used in this thesis are as follows: G-1-P = glucose-1-phosphate; G-6-P = glucose-6-phosphate; F-6-P = fructose-6-phosphate; UDP = uridine diphosphate; UTP = uridine triphosphate; NAD = nicotinomide adenine dinucleotide; FAD = flavin adenine dinucleotide; ATP = adenosine triphosphate; ADP = adenosine diphosphate; AMP = adenosine monophosphate; TCA = trichloroacetic acid; EDTA = ethylenediamine tetraacetate; LDH = lactic dehydrogenase; glycogen synthetase = uridine diphosphoglucose-glycogen-transglucylase; N_aF = sodium fluoroacetate; PPO = 2, 5-diphenyloxazole; POPOP = 1, 3-Bis 2-(5 phenyloxazolyl)-benzene; MVO_2 = maximal oxygen uptake.

produced is the direct source of energy for muscular contraction.

Glycogen is synthesized from G-6-P by an enzyme called glycogen synthetase. This enzyme exists in two interconvertible forms: synthetase I, which is active in the absence of G-6-P and synthetase D, which is G-6-P dependent. In living muscle, the relative amounts of these two forms of glycogen synthetase are related to the tissue concentration of glycogen. There is speculation that glycogen controls its own synthesis through a feedback mechanism which stimulates the interconversion of glycogen synthetase (I⟷D) in such a way as to favour glycogenesis when tissue concentrations are low and to retard synthesis when tissue concentrations are high (21).

Many investigators have demonstrated that exercise and training increases enzymatic activity in muscle. Danforth (21) has reported greater glycogen synthetase activity in trained muscle when compared with untrained muscle. Jeffress et al (33) demonstrated an increase in the total glycogen synthetase activity in red and white skeletal muscle with training. Barnard and Peter (7) and Peter et al (50), working with small animals, found that adaptation to exercise occurred through increased activities of enzyme systems. The muscle biopsy technique developed in Sweden by Bergstrom (11), has enabled researchers to study the biochemical adaptations of human muscle to exercise and training.

Rationale behind the Study

Several investigators have demonstrated the influence of exercise and training on muscle glycogen metabolism in humans (3,4, 12, 40). Saltin and Hermansen (55) and Ahlborg et al (4) reported that the local glycogen store in the working muscle is a determining factor in the performance of long term work. Lappage (40) investigated the effect of exercise and training in muscle glycogen levels in humans. The results indicated that the muscle glycogen stores of trained individuals were higher than those of non-trained individuals.

Bergstrom et al (13) and Lamb (38) reported a supercompensation effect of glycogen following exercise which appeared to be associated with increased skeletal muscle enzyme activity. Jeffress et al (33) and Lamb et al (39) in their work with animals have reported an increase in the activity of the enzymes directly related to glycogen synthesis as a result of exercise and training. However, the effect of exercise and training on glycogen synthetase in humans has not been reported.

A study was necessary to determine the effect of exercise and training on glycogen synthetase activity in man. An investigation of this nature might explain the supercompensation effect of glycogen as well as the depletion of glycogen during prolonged and short term exercise.

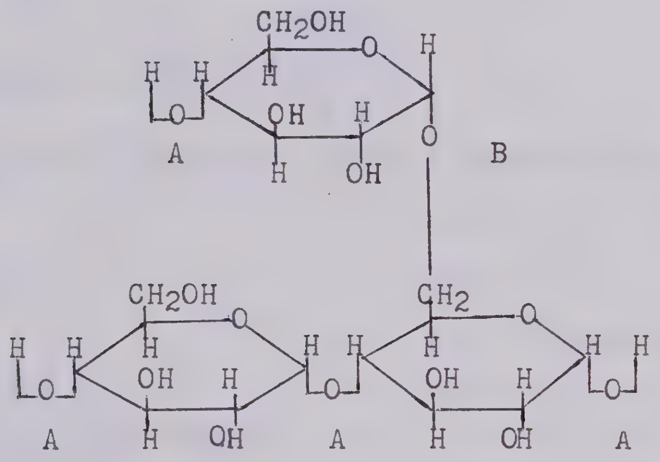
The enzymatic adaptations which occur during and after prolonged exercise, maximal exercise and the effects of a training regimen on enzymatic activity need to be investigated.

CHAPTER II

REVIEW OF LITERATURE

Glycogen, a highly branched polysaccharide, consists of repeating units of glucose in α1,4 and α1,6 linkage. These are illustrated in the following diagram.

Glucose and α1,4 α1,6 Linkage (65)



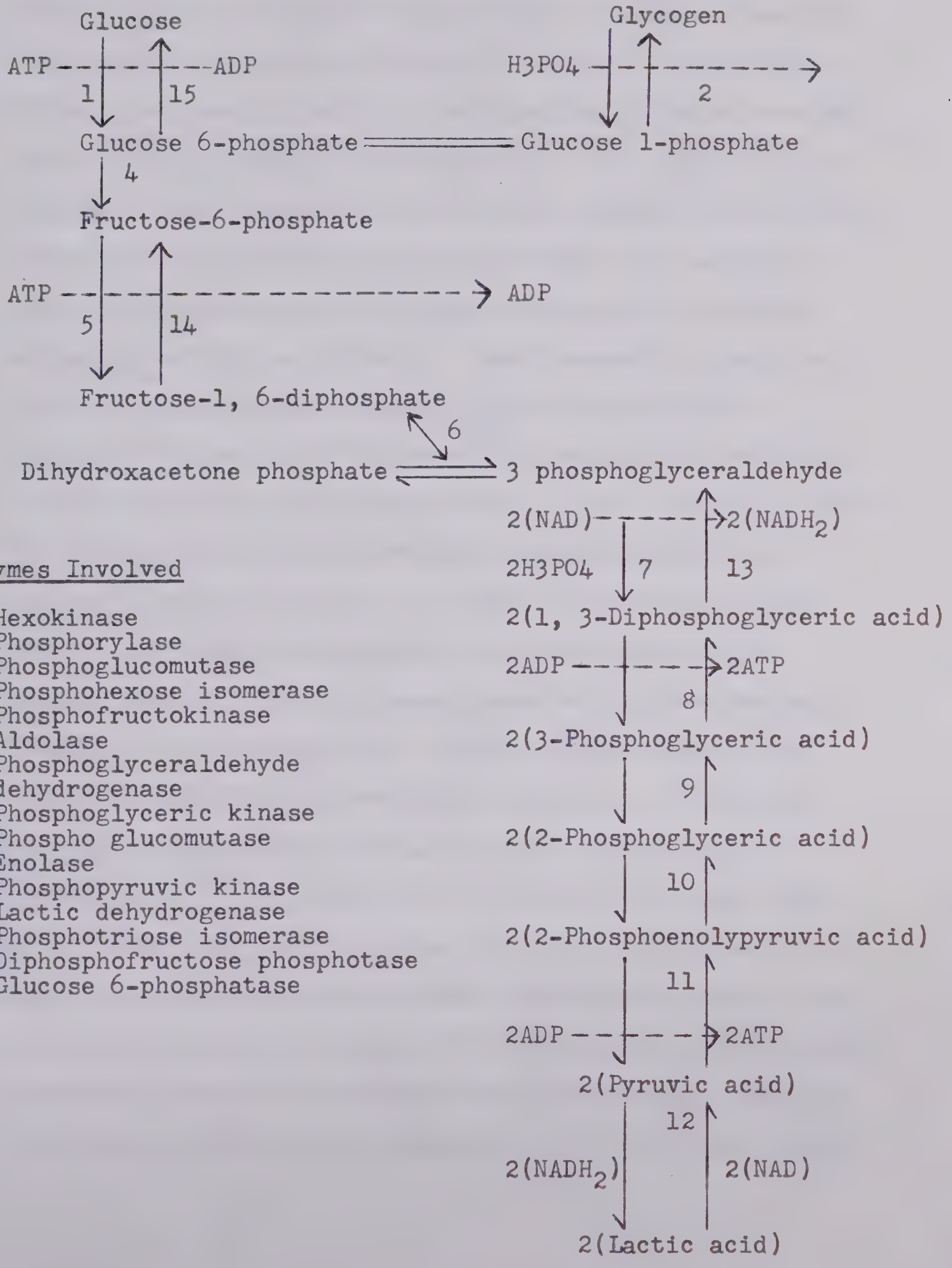
A = α, 1,4-glucosidic bond

B = α, 1,6-glucosidic bond

The Glycolytic Pathway

The mechanical energy required for muscular activity is provided by the hydrolysis of ATP to ADP and Pi. This breakdown of ATP during muscular contraction was demonstrated in 1962 by Cain and Davies (16). During muscular exercise,

THE GLYCOLYTIC PATHWAY (26:17)



Enzymes Involved

1	Hexokinase
2	Phosphorylase
3	Phosphoglucomutase
4	Phosphohexose isomerase
5	Phosphofructokinase
6	Aldolase
7	Phosphoglyceraldehyde dehydrogenase
8	Phosphoglyceric kinase
9	Phospho glucomutase
10	Enolase
11	Phosphopyruvic kinase
12	Lactic dehydrogenase
13	Phosphotriose isomerase
14	Diphosphofructose phosphotase
15	Glucose 6-phosphatase

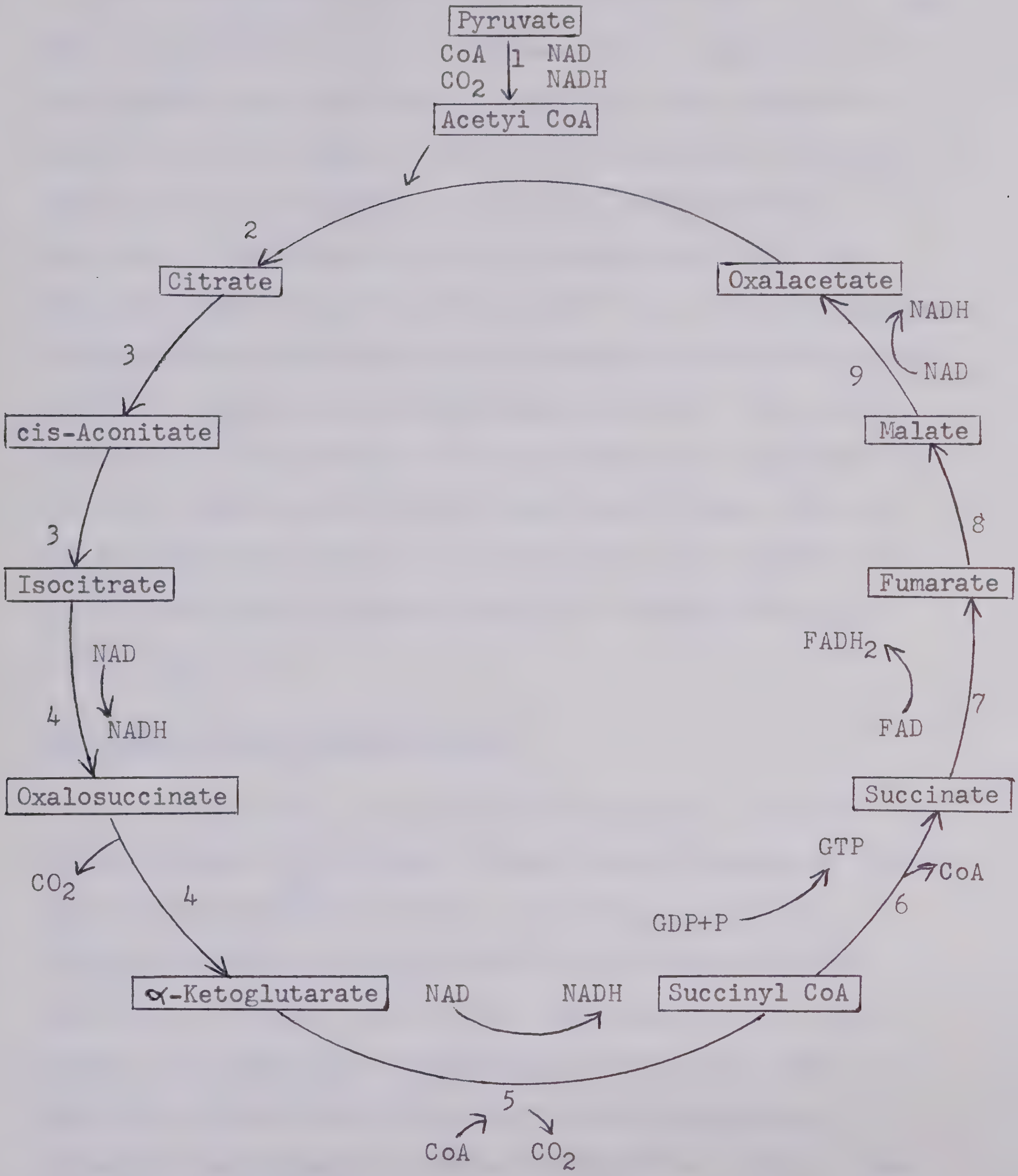
the biochemical processes of the muscles must provide a continuous supply of ATP in order to support the process of muscle contraction as well as to drive endergonic reactions which require an outside source of energy. Synthesis of ATP may be accomplished via the Citric Acid Cycle provided there is an adequate supply of oxygen. Synthesis of ATP without oxygen (anaerobic glycolysis) utilizes carbohydrate exclusively whereas the synthesis of ATP under aerobic conditions can derive its initial substrate from either carbohydrate, fat or protein. Under anaerobic conditions the net yield of usable energy may be computed from a balance of moles of ATP consumed and regenerated per mole of glucose degraded to lactic acid. In this case one mole of ATP is consumed in phosphorylation of glucose to G-6-P. Glucose is phosphorylated by enzymes called hexokinases which occur almost universally in living cells (15). Another mole of ATP is consumed when F-6-P is converted to fructose-1, 6-diphosphate. A total of four moles of ATP are regenerated by the reactions in which 1, 3-diphosphoglyceric acid and phosphoenal pyruvate are converted to pyruvic acid. Thus, per mole of glucose glycolyzed, two moles of ATP are generated for a net gain of two moles of ATP. Under anaerobic conditions, approximately 30% of the potentially available energy of the conversion of glucose to lactic acid may be stored in the form of ATP. Most of the energy is lost to the system as heat. Although the net

yield of ATP is small under anaerobic conditions, the lactic acid produced can provide energy when the accumulated lactic acid is metabolized under aerobic conditions. Glycogenesis from lactate does not occur to any appreciable extent in muscle, but due to the fact that lactate can freely diffuse through the cell wall, it is carried by the blood to the liver where glycogenesis occurs (10). This mechanism is important, as an accumulation of lactate in the muscle would disturb the equilibrium of the overall reaction scheme. Equilibrium favours the degradation of glucose to pyruvate or lactate but the accumulation of any intermediate compound shifts the reaction back towards glucose. This shift is important for the regeneration of NAD+ which functions as an electron acceptor in the glycolytic chain. The accumulation of the reduced form (NADH) which would occur, provided the pyruvate diffused from the cell prior to its' reduction to lactate, would result in a stoppage of the glycolytic pathway with the formation of glyceraldehyde-3-phosphate.

Citric Acid Cycle

When a sufficient supply of oxygen is available, pyruvic acid is oxidatively decarboxylated to acetyl CoA and CO_2 (18). This occurs in the mitochondria of the cell whereas glycolysis takes place in the cytoplasm. Complete oxidation of each mole of acetyl CoA yields twelve moles of

THE KREBS CYCLE (TRICARBOXYLIC ACID CYCLE) (29)



Enzymes Involved

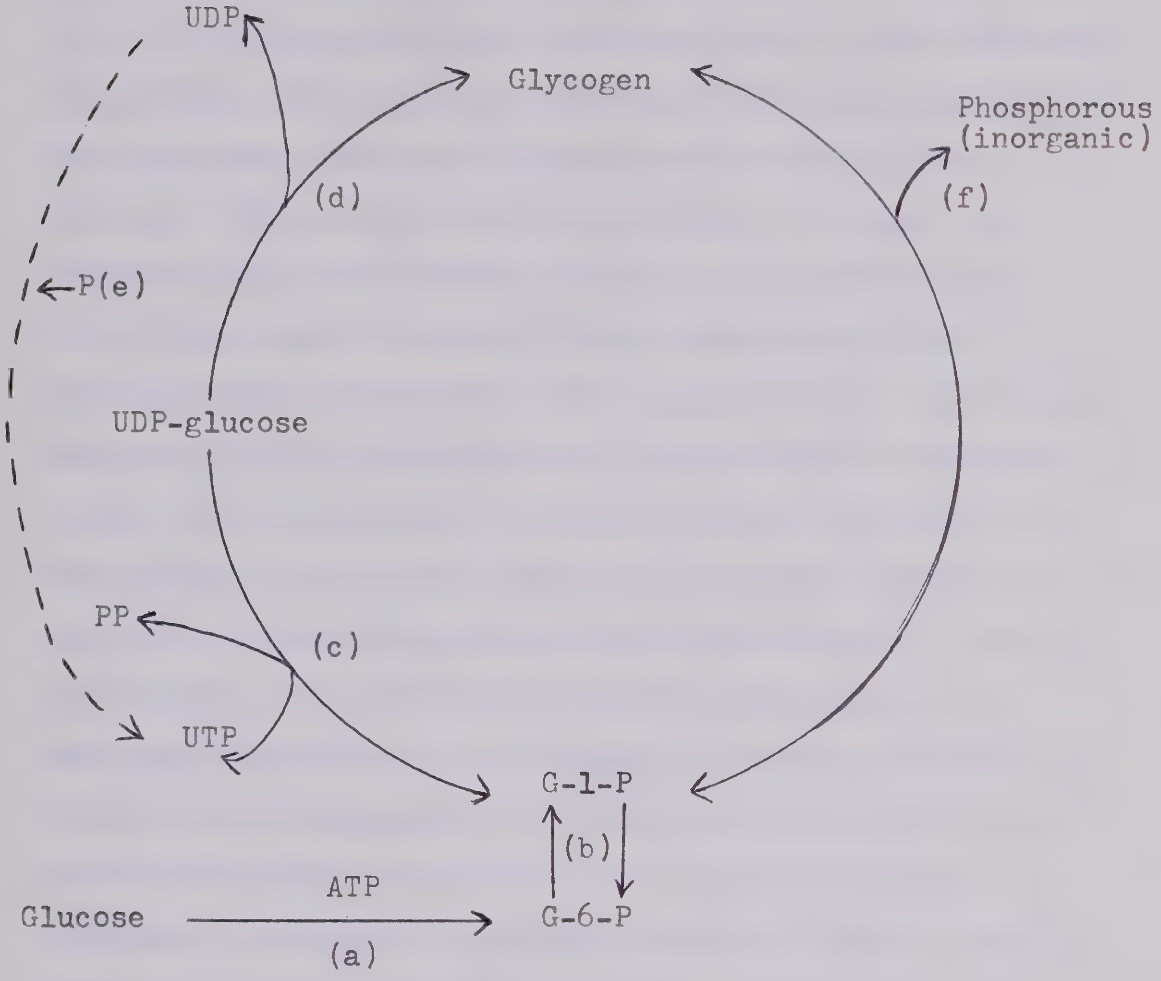
1 Pyruvic dehydrogenase
2 Condensing enzyme
3 Aconitase
4 Isocitric enzyme
5 α-Ketoglutarate dehydrogenase
6 Succinic thiokinase
7 Succinic dehydrogenase
8 Fumarase
9 Malic dehydrogenase

ATP or twenty-four moles of ATP per glucose equivalent via the Electron Transport Chain. Also, for each revolution of the cycle, one mole of acetyl CoA is consumed and two moles of CO_2 are evolved. Oxaloacetic acid which is utilized in the initial step, is regenerated. The most important function of the Citric Acid Cycle is the oxidation of substrates in the cycle by the removal of electrons which result in the reduction of hydrogen carriers. The initial acceptors of the hydrogen ions are NAD^+ and a flavo protein, FAD (46). Each mole of NADH gives rise to three moles of ATP whereas $FADH_2$ gives rise to only two moles of ATP due to the fact that the latter occurs at a higher oxidative level (65).

Glycogenesis and Glycogenolysis

Glycogenesis, an intercellular process, involves the active transport of glucose through the cell membrane from the extracellular fluid (10). The hormone, insulin, facilitates this active transport by increasing the permeability of the cell wall (20). Upon entering the cell, glucose is immediately phosphorylated to G-6-P. This reaction is essentially irreversible with equilibrium favouring the formation of G-6-P by 6,300 to 1 (65). However, an enzyme, glucose-6-phosphotase, catalyzes the hydrolysis of G-6-P to glucose and Pi. By this mechanism, glucose is released into the blood from the liver. The

GLYCOGEN SYNTHESIS AND BREAKDOWN (60)



Pathways of the synthesis and breakdown of the ∝ - 1, 4' linkages of glycogen (60).

The solid arrow heads indicate the probable preponderent direction of the reactions.

a - hexokinase
b - phosphoglucomutase
c - UDPG-pyrophosphorylase
d - glycogen synthetase and branching enzyme
e - UDP-kinase
f - phosphorylase

reaction does not take place in muscle as the enzyme, glucose-6-phosphotase is not present (18).

G-6-P appears to act as an activator in the synthesis of glycogen (15). Manners (47) found that G-6-P seemed to increase the affinity of glycogen synthetase for UDP-glucose. This would agree with the work of Leloir and Goldemberg (42) who observed that with the addition of G-6-P there was an increase in the formation of UDP-glucose. Traut and Lipmann (61) suggested that the increase was due to G-6-P protecting the enzyme from inactivation. Anthony et al (5) implied that G-6-P acted by binding to the glycogen synthetase, reshaping the enzyme, with a resulting increase in affinity for the substrate. G-6-P, once formed, was converted to G-1-P by the action of phosphoglucomutase in the presence of catalytic amounts of glucose 1, 6-diphosphate. The G-1-P then reacted with UTP to form an active nucleotide. This is the immediate precursor of glycogen. Glycogen cannot be formed from UDP-glucose alone as there must be present an acceptor poly-glucose chain which, in muscle and liver, is glycogen, itself (47). Leloir and Goldemberg (42) found that the best primer for the reaction was glycogen and that enzymatic activity decreased following degradation of glycogen. Luck (45) revealed that the enzyme which catalyzed the formation of glycogen from UDP-glucose was bound to glycogen and not to liver cell structures. The synthesis

of the glycogen chain was catalyzed by glycogen synthetase which results in the irreversible transfer of an α-D-glucose residue from UDP-glucose to an acceptor containing α1,4 linked D-glucose residues (47). Glycogen is a highly branched structure made up of glucose residues in α1,4 and α1,6 bonds.

Glycogen synthetase catalyzes only the formation of α1,4 bonds. Therefore a second enzyme, amylo (1,4 $\longrightarrow$ 1,6)-transglucosylase is necessary to catalyze the formation of branch points at α 1,6 bonds at a frequency of approximately every eight to twelve glucose units (64). When there is a demand for glucose or increased energy demands, which occurs during exercise, glycogen is degraded to G-1-P by the action of phosphorylase. The G-1-P is then converted to G-6-P which can then be converted to glucose in the liver via the Cori Cycle. The overall pathway for glycogen synthesis and degradation does not utilize ATP nor does it produce ATP in the breakdown to G-6-P.

Effect of Exercise and Training on Glycogen Metabolism

Wilbur (66), experimenting with mice which were forced to swim to exhaustion, found that muscle glycogen level dropped sharply during the first twenty minutes of swimming. Thereafter it levelled off and showed no further measurable change. The correlation between muscle glycogen content and swimming time was extremely low. Poland and

Blount (51) found a decrease in myocardial glycogen levels in male Sprague-Dawley rats following graded exercise. Yampolskaya (69) demonstrated with rats that a brief intensive effort was characterized mainly by expenditure of muscle glycogen. However, after a substantial rest period, a significant resynthesis of muscle glycogen occurred.

Poland and Blount (52) demonstrated that a trichloroacetic acid soluble fraction of cardiac glycogen was higher in the trained group of rats following a twenty-four hour fast. However, following a fifteen minute run the TCA glycogen content of the trained hearts was not significantly different from that of the non-trained hearts. This could be explained by the studies which showed that hypoxia and work load are important factors in determining myocardial glycogenolysis during exercise. The mild training may not have relieved the hypoxia which was produced during the exercise (52). Lamb (38), experimenting with guinea pigs for the purpose of clarifying the physiological mechanism responsible for the exercise-induced supercompensation of glycogen, noted that an acute bout of exercise seemed nearly as effective as chronic exercise in stimulating glycogen supercompensation. Fourty-eight hours after the thirty minute exercise bout, the untrained group showed a muscle glycogen content almost as high as the trained group. The untrained group depleted

their glycogen muscle stores after only thirty minutes of exercise whereas the trained group depleted their glycogen stores after eighty minutes. Proctor and Best (53), in a classical study of muscle glycogen, trained healthy dogs to run on three legs. The dogs were exercised for two to three weeks and the rectus femoris muscle of the sedentary and the exercised legs compared for glycogen content. The results suggested that there may be an optimum period of training for the accumulation of glycogen. Muscles trained longer than the optimum period retained their increased ability to perform work but did not demonstrate an extra increase in glycogen levels. The results of experiments with trained rats by Yampolskaya (69) suggested that the use of brief periods of training, three to thirty times shorter than maximal training periods, resulted in considerably smaller losses of creatine phosphate and glycogen in trained animals than in untrained animals following exercise. Grollman (28), investigating the effects of training on glycogen levels in rats, showed that the trained group had greater muscle glycogen values than the untrained group prior to their final bout of exhaustive exercise. These results indicated that the increased stores of glycogen stemming from training might be useful in extending muscular endurance during exercise.

The muscle biopsy technique developed in Scandinavia has enabled researchers to discover the effects of exercise

upon human muscle glycogen (11). Research has shown that during exercise there is continuous utilization of muscle glycogen (3, 4, 12). Bergstrom and Hultman (13) demonstrated that this muscle glycogen utilization was confined to the working muscle. Each subject pedalled with one leg while the other rested. Following exercise, the exercised leg had very low glycogen stores as compared with the rested leg. Three days following the exhaustive exercise, the exercised leg had two times the glycogen level of the rested leg. They concluded from the data that exercise resulting in glycogen depletion enhanced the resynthesis of glycogen. Saltin and Hermansen (55) and Ahlborg et al (4) concluded from their results that the local glycogen stores in the working muscles were a determining factor in the ability to perform long term work.

Several investigators have reported that during prolonged exercise, blood glucose levels decrease below that of the resting value but not to such an extent as to be the determining factor with respect to fatigue (4, 59). In fact, Ahlborg et al (4) performed an experiment in which two subjects were administered 330g. and 180g. of glucose while exercising. The results indicated that the lowering of glycogen was of the same order as when subjects were exercised without administration of glucose despite the fact that the glucose content in the arterial blood was greatly increased. This suggested that the glycogen

catabolism during exercise takes place at almost the same rate, irrespective of the quantity of glucose delivered to the muscle.

Saltin and Hermansen (55) and Ahlborg et al (3) investigated the effect of diet on glycogen stores and the ability to perform prolonged exercise. The subjects were exposed to a mixed diet, a fat diet, a protein diet and a carbohydrate diet. On a fat and protein diet, the body was only able to synthesize muscle glycogen very slowly following glycogen depletion. On a diet rich in carbohydrates, a vigorous resynthesis took place resulting in very high muscle glycogen values following exercise. Prolonged glycogen deficiency may give rise to an enzymatic adaptation which leads to a rapid resynthesis of glycogen at supernormal levels.

Enzymatic Adaptations to Exercise and Training

Barnard and Peter (7) and Peter et al (50), working with small animals, found that adaptation to exercise occurs through increased activities of enzyme systems. Holloszy (32) investigated the effects of exercise on respiratory enzyme activity in skeletal muscle. The results indicated that the exercised animals exhibited a high degree of respiratory control and tightly coupled oxidative phosphorylation. Thus the increase in electron transport capacity was associated with a concomtiant rise in the

capacity to produce ATP. The enzymatic adaptation that occurred as a result of exercise was a two-fold increase in the enzymes of the electron transport chain. As well, the concentration of cytochrome C doubled, giving evidence that the rise in enzyme activity was due to a net increase in enzyme protein. This adaptation may partially account for the increase in aerobic work capacity that occurred with regularly performed, prolonged exercise.

Cori (19), examining the connection between mechanical work of contracting muscle and the speed of chemical reactions which furnish the necessary energy, showed that compared to rest, glycogenolysis may be speeded up several hundred times by single twitches and up to a thousand times by tetanus. Following contraction, the enzymatic processes quickly returned to the resting rate. Five seconds following tetanus, the glycogen breakdown had decreased from 400mg/100g (wet muscle)/minute to 3mg/100g/minute. Jones and Perry (34), in results obtained with mammalian and insect skeletal muscles in vivo and in vitro, concluded that continued contractile activity led to an increase in the activity of the enzymes associated with muscle metabolism. Gollnick et al (27) found that exercise did not produce any significant change in LDH activity of skeletal muscle. The lack of increase in the skeletal LDH activity may be related to the fact that the work load of the training program was aerobic in nature.

Several researchers have shown an increase in serum enzyme values in rats as a result of exercise and training (6, 24, 31). Altland and Highman (6) found a two to six-fold increase in serum glutamic oxalacetic transaminase, serum glutamic pyruvic transaminase, serum lactic dehydrogenase and serum adolase immediately after exercise. Garbres et al (24) have shown a marked increase in serum enzymes after sixteen hours of exercise in untrained rats. The rise in serum enzymes may perhaps be best explained by alterations in cellular permeability.

Glycogen Synthetase

The synthesis of the amylase chain of glycogen is catalyzed by glycogen synthetase, an enzyme that catalyzes the following reaction:

$$n\ \text{UDP-glucose} \longrightarrow (\text{glucose})n + n\ \text{UDP}$$

Leloir et al (43), working with rat muscle preparations, determined that the best primer for glycogen synthetase activity was glycogen. In a later study in liver glycogen synthetase, Leloir (42) found that with centrifugation of liver extracts, glycogen synthetase appeared to be bound to the glycogen as an enzyme substrate complex.

A number of researchers have established that glycogen synthetase exists in two forms (I + D) (2, 20, 25, 54). Danforth (20) concluded that glycogen controls its own synthesis by a mechanism which affects the inter-

conversion of glycogen synthetase I$\longleftrightarrow$D in such a manner as to favour glycogen synthesis when tissue concentrations are low and to slow synthesis when tissue concentrations are high. Glinsman and Hern (25) deduced that the $3^{1}5^{1}$-cyclic phosphate esters of adenosine, quanisine, cytodine and uridine enhance the conversion of glycogen synthetase I to D in rat liver. Glycogen synthetase activity in liver is thought to be controlled by a phosphorylation-dephosphorylation sequence. An active dephosphorylated I form of the enzyme is converted to an inactive D form by a kinase reaction requiring ATP and Mg++. The $3^{1}5^{1}$-cyclic nucleotides may have direct action on a kinase which converts glycogen synthetase I to D. In liver it is likely that a decrease in glycogen synthetase I activity is secondary to activation of glycogen synthetase I kinase system. Vardanis (63) looked into the transformations of the enzyme in vitro from the liver tissue of mice. The results pointed to the existence of a third form of the enzyme that is inactive even in the presence of G-6-P. Also, the complexing of native glycogen with concanavalin A completely prevents transformations of the bound synthetase. Perez (54), examining the two forms of the enzyme in dog skeletal muscle, determined that the enzyme extracted from fresh muscle exhibited a dependence for G-6-P. A sulfhydryl reagent increased the conversion of the D to I form.

The conversion of the I→D was achieved by adding ATP and Mg++ which operated only in the absence of G-6-P. The addition of G-6-P resulted in a parallel decrease in the activity of the I→D conversion. This suggests the possibility of a scheme of interconversion through an inactive intermediate in the I↔D conversion. Mersmann and Segal (48) contested the existence of the I and D forms in liver and proposed a form that was inactive in vivo (D-form) and which was converted in vitro to the active I form. The difference in the two forms was not in the absolute requirement for G-6-P but rather a higher affinity for UDP-glucose, G-6-P and glycogen by the I-form. Other researchers have demonstrated that the synthesis of glycogen in vivo by glucose correlated well within vitro measurements of glycogen synthetase activity when performed in a concentrated homogenate. The stimulation was, in a way, related to the intercellular levels of G-6-P. Liver glycogen synthesis and synthetase activity are subject to control by an undetermined mechanism.

Traut and Lipmann (61) attempted to determine the mechanism of activation of glycogen synthetase by G-6-P. The G-6-P exerted its stimulating effect through an increase in the affinity of the enzyme for UDP-glucose. It was inferred that the activator, G-6-P, had a special binding site on glycogen synthetase, reshaping the enzyme with a resulting increase in the affinity for the substrate.

Steiner and King (59) illustrated the structural requirements for G-6-P exhibited by glycogen synthetase. The phosphate group at position 6, the OH or amino substituent in the proper orientation at position 2 and the five carbon heterocyclic ring structure were all necessary for stimulation of glycogen synthetase. The stimulating mechanism was operated by the decreased binding affinity for UDP in the presence of G-6-P. Thus, G-6-P decreased the inhibitory affect of UDP in a partially competitive manner. Kornfield and Brown (36) showed that the reaction was activated by addition of Mg++ and G-6-P. The extent of stimulation by Mg++ depended on the concentration of UDP-glucose and on the presence or absence of G-6-P. Leloir et al (43) found that hexose-6-phosphates activated the reaction by increased action of the glycogen forming enzyme. They also ascertained that for each mole of UDP-glucose one mole of UDP was formed and one mole of glucose was added to the glycogen chain.

A number of investigators have shown the effect of fasting on glycogen synthetase activity. Belford and Cunningham (8) determined that periods of starvation of eighteen, forty-eight and ninety-six hours progressively lowered the total (I + D) and the I-form of glycogen synthetase in the rat heart. In contrast, no significant changes in the skeletal muscle occurred after the same periods of starvation. Adroundy (2), in a similar study,

found that while the total of I + D activity of the heart stays unaffected by fasting, the I-form undergoes a significant decrease with a corresponding increase in the D-form. While a similar tendency may be detected in the case of skeletal muscle, the data did not allow for a conclusive statement.

Many researchers have studied the effect of hormones on glycogen synthetase activity (9, 20, 35, 49, 59). Palasi and Larner (49) have indicated that glycogen synthetase activity was increased in extracts of diaphragm incubated with insulin when assayed without G-6-P. When G-6-P was added to the assay, the activity of extracts prepared from control and insulin treated diaphragm was increased and identical. This tended to demonstrate that the increase in activity could be due to an increased amount of a cofactor which alters the protein structure or binds at a particular enzyme site responsible for the insulin treated glycogen synthesis. Steiner and King (59) concluded that the increase in glycogen synthetase activity, after the administration of insulin, was due to accelerated synthesis of enzyme protein. The acceleration of enzyme synthesis can be inhibited by actinomycin D. This fact offered strong evidence for the increase of enzyme protein synthesis with insulin treatment. Belicopitow (9) examining the effects of epinephrine on glycogen synthetase, illustrated that it exerted an effect on more than one

enzyme. Epinephrine resulted in a decrease in glycogen synthetase activity which was due to increased phosphorylase activity with epinephrine administration. Increased phosphorylase activity may affect glycogen synthetase activity by decreasing the stability of synthetase. This would be caused by a gradual degradation of glycogen by successive removal of newly added glucose residues. Karpatkin et al (35) in accordance with Belicopitow found that administration of epinephrine resulted in stimulation of phosphorylase, leading to glycogen degradation.

DeWulf and Hers (22) determined that an intravenous administration of glucose in rats stimulated a fifteen to forty fold increase in the conversion of glucose to glycogen. During the period of glycogen synthesis, the concentration of hepatic G-6-P and UDP-Glucose was markedly lower whereas the activity of glycogen synthetase was greatly elevated.

Effects of Exercise and Training on Glycogen Synthetase

Adaptation of skeletal muscle to vigorous exercise may occur through increased activities of enzyme systems providing energy for muscular contraction. Such a response has been clearly demonstrated for creatine phosphokinase (34), hexokinase (50) and several oxidative enzymes (32).

Jeffress et al (33) measured the activities of the G-6-P independent I-form and dependent D-form of glycogen

synthetase in red and white skeletal muscle and compared the effects of single and repeated bouts of exercise. Trained guinea pigs exhibited increases in the (I + D) enzyme activity in red and white skeletal muscle, as well as an increase in the amount of activity in the I-form. Also, glycogen synthetase (I + D) activity was greater in red skeletal muscle than in white. In a similar study by Lamb et al (39), the guinea pigs were divided into six groups according to the nature of their exercise program. Exercise decreased the glycogen stores of red and white skeletal muscle. Forty-eight hours following exercise, skeletal muscle demonstrated a supercompensation of glycogen up to seventy-five percent greater than the resting values. The total glycogen synthetase activity of heart muscle of exercised animals forty-eight hours after running was greater than that of the sedentary control groups. There were no significant changes in the activity of liver glycogen synthetase. Increased skeletal muscle enzyme activities appeared to be associated with glycogen supercompensation.

Staneloni and Piras (58) demonstrated that electrical stimulation *in situ* of rat skeletal muscle resulted in an I to D conversion which took place within the first five seconds of stimulation. During the recovery period, glycogen synthetase reverted to the more active I form. Canal and Frattola (17) exercised rats almost to the point

of exhaustion and at a given time, following exercise, analyzed the gastronemius muscle for glycogen synthetase. There was a marked increase of glycogen synthetase in both the I and D form at all time intervals measured.

CHAPTER III

METHODS AND PROCEDURES

A group of eighteen male subjects ranging in age from eighteen to twenty-eight years were divided into two groups: sedentary and active. The active group underwent training in preparation for competition in track or wrestling. The track training program lasted five months and included weight training and hour long practices five times per week as well as regularly scheduled competitions. The wrestlers' training program lasted five months and included various running programs, weight training and hour long practices five times per week as well as regular competition. The sedentary group did not participate in activity on a regular basis.

During the week prior to the submaximal test session and before the training program had begun, each individual was tested for MVO_2 on a bicycle ergometer. The MVO_2 was determined using the Astrand Modified Bicycle Ergometer Test as further modified by Shephard (56). All subjects underwent both the submaximal and maximal tests, during which muscle samples were taken in early November of 1969. The test sessions were from 4:00 to 7:00 p.m. Monday and Thursday and from 9:00 and 12:00 noon on Saturday. The active group was tested in November prior to their competitive season and then retested five months later immediately

following their competitive season. Permission to participate in the study was obtained from each subject prior to the initial test session.

No effort was made to control the diet and activity of the subjects prior to testing except they were asked to refrain from very strenuous exercise or abnormal diets for one day prior to testing. They were also asked to abstain from alcohol for twenty-four hours prior to the test. The subjects participated in the submaximal test first.

Muscle samples were taken from the vastus lateralis at various times before, during and after the submaximal test. The biopsy needle consisted of a sharp-edged aluminum cylinder which fit into a hollow outer cannula (3.5 - 5.0mm in diameter) with a window-like opening being near the tip (11).

Several minutes after a local anaesthetic (xylocaine hydrochloride) was injected into the area of the vastus lateralis, an incision of approximately 1cm was made with a scalpel. The biopsy needle was then advanced 4 - 5cm into the muscle. The cylinder was pulled back a few centimeters while the cannula remained in place. It was then pushed back again, severing off a small piece of muscle (approximately 30 - 80mg) which protruded into the window of the needle. The muscle sample was then carefully transferred to the weighing pan where all visible

fat and connective tissue were removed using forceps. The sample was then weighed using a Roller-Smith Precision Balance and transferred with forceps to an appropriately labelled test tube. A probe was then used to push the sample to the bottom of the capped test tube which in turn was frozen as quickly as possible in dry ice and 95% ethyl alcohol. The pan, with traces of blood was reweighed and this weight was subtracted from the initial weight of the muscle sample.

Initially, three biopsies were taken from the resting subject's right or left thigh. At the same time the subject was connected to an infra-venous drip to allow for removal of blood from the brachial vein. The blood was analyzed for glucose (37) and lactate (57) content. The subject, with chest electrodes in place, mounted a Monark bicycle ergometer and commenced pedalling at a work load corresponding to 70% MVO_2. The heart rate of the subject was monitored at five minute intervals throughout the test. After every twenty minutes of exercise the subject stopped momentarily so that a biopsy sample could be taken and blood withdrawn. The submaximal test was completed when the subject failed to pedal at the required rate. A muscle sample as well as a blood sample was immediately taken at the time of exhaustion. Ten minutes following cessation of exercise a further sample of muscle and blood were taken.

Fourteen days following the submaximal test, each subject was required to perform a maximal test which corresponded to a work load of 100% MVO_2. In the maximal test, muscle from the vastus lateralis and blood from the brachial vein were removed at rest, fatigue and ten minutes after cessation of exercise. Heart rate was monitored continuously throughout the exercise bout.

With completion of the training program in March, 1970, all physiological measurements were taken for the active group in a similar fashion to the pre-training tests. Thus, the MVO_2 was again determined for each individual comprising the active or trained group. Following this, each subject was required to repeat the submaximal and maximal tests, during which, muscle biopsies and blood samples were taken. The muscle samples were analyzed for glycogen content and glycogen synthetase activity at the Surgical Medical Research Institute Laboratory. Muscle glycogen was analyzed by the method of Lo et al (44).

Human muscle samples from the vastus lateralis were analyzed for glycogen synthetase activity in the following manner. The samples (30 - 80mgm) were frozen with liquid nitrogen in 4ml of buffer solution containing 20mm sodium glycerophosphate, 1mm ETDA, 100mm N_aF and 20mm mercatoethanol, ph 6.10 ± 0.05. The frozen buffer and sample were ground to a fine paste in liquid nitrogen using a mortar and pestle and then allowed to thaw. The resulting

suspension was centrifuged for five minutes at 1500g and the supernatant used for the assay. The extracts were kept in ice at all times.

For the assay procedure, tubes were prepared containing 0.1ml of the substrate solutions. These were equilibriated for fifteen minutes together with the muscle extract samples in a $30^{\circ}C$ water bath. One-tenth ml samples were then added to the substrate tubes, shaken and timed. Reaction was normally allowed to proceed for fifteen minutes in the synthetase measurements. The reactions were stopped by addition of 1ml of an ice cold solution containing 6% TCA, 2mg LiBr, and 1mg of glycogen. The proteins were removed by centrifugation at 1000g for ten minutes and the supernatant was decanted. Two ml of cold 95% ethanol were added and the samples allowed to sit for twenty minutes to precipitate the glycogen. The samples were then centrifuged at 2000g for ten minutes, the supernatant decanted and the precipitates washed with 66% ethanol and recentrifuged twice. The glycogen pellets were finally resuspended in 1ml of ethanol and washed into counting vials with 1ml of water. The vials were taken to dryness in a vacuum oven with minimal heat. One ml of NCS reagent (Nuclear Chicago Corporation) was added and the vial well shaken. Fifteen ml of toluene containing 4gm/l of PPO (Beckman Instruments Inc.) and 50mgm/l of POPOP (Beckman Instruments Inc.) were added and the samples were counted

in a Nuclear Chicago Mk 1 liquid scintillation counter. Counting efficiencies were determined by the channels ratio method. Blank samples were run adding the sample after the TCA quenching solution.

The substrate solutions were as follows:

Synthetase:

Solution I:

1% glycogen, 1mM UDPG C^{14}(4.3×10^8 dpm/mM) (New England Nuclear Corp., Sigma Chemical Co.) 5 mM Tris HCl, pH 8.2.

Solution D:

As above, plus 10 mM G-6-P (Sigma Chemical Co.).

For the human muscle samples, higher specific activities were required and solutions of UDPG of 1.32×10^9 dpm/mM and G-6-P of 4.3×10^7 dpm/mM were used.

A two-way analysis of variance (67) with repeated measures on factor B (Pre vs. Post) and factor C (Treatments) was run to test the variance between group means and between treatment means. The model was also designed to test for interaction between groups. A two-way analysis of variance with repeated measures in one factor (B = treatments) was utilized to test the variance existing between the sedentary and post-training groups. Multiple comparisons were made using Tukey's W Procedure (67) to establish significant differences between treatment means. Lastly, tests on overall differences were run. The follow-

ing comparisons were made: Initial with Fatigue, Initial with Recovery and Fatigue with Recovery.

CHAPTER IV

RESULTS

Figures 1, 2, 3 and 4 represent the glycogen synthetase activity for each group on the submaximal and maximal test. The mean initial, fatigue and recovery samples were considered biologically equivalent despite the time variables between individuals. Each graph that was plotted represented the mean glycogen synthetase activity and standard error of the mean for a particular treatment.

Figure 1 illustrates the histographs of glycogen synthetase D activity for each group on the submaximal test. The graphs represented a decline in glycogen synthetase D activity from initial to fatigue. During the ten minute recovery period, both the trained and sedentary group exhibited an increase in synthetase D activity above the fatigue levels.

In the maximal test (Fig. 2) both the trained group and the pre-trained group experienced a small decline in synthetase D activity from initial to fatigue. However, the sedentary group demonstrated a dramatic decrease in synthetase D activity from initial to fatigue. During the ten minute recovery period, the trained and sedentary groups exhibited a substantial increase in synthetase D values.

In the submaximal (Fig. 3) and maximal (Fig. 4) tests,

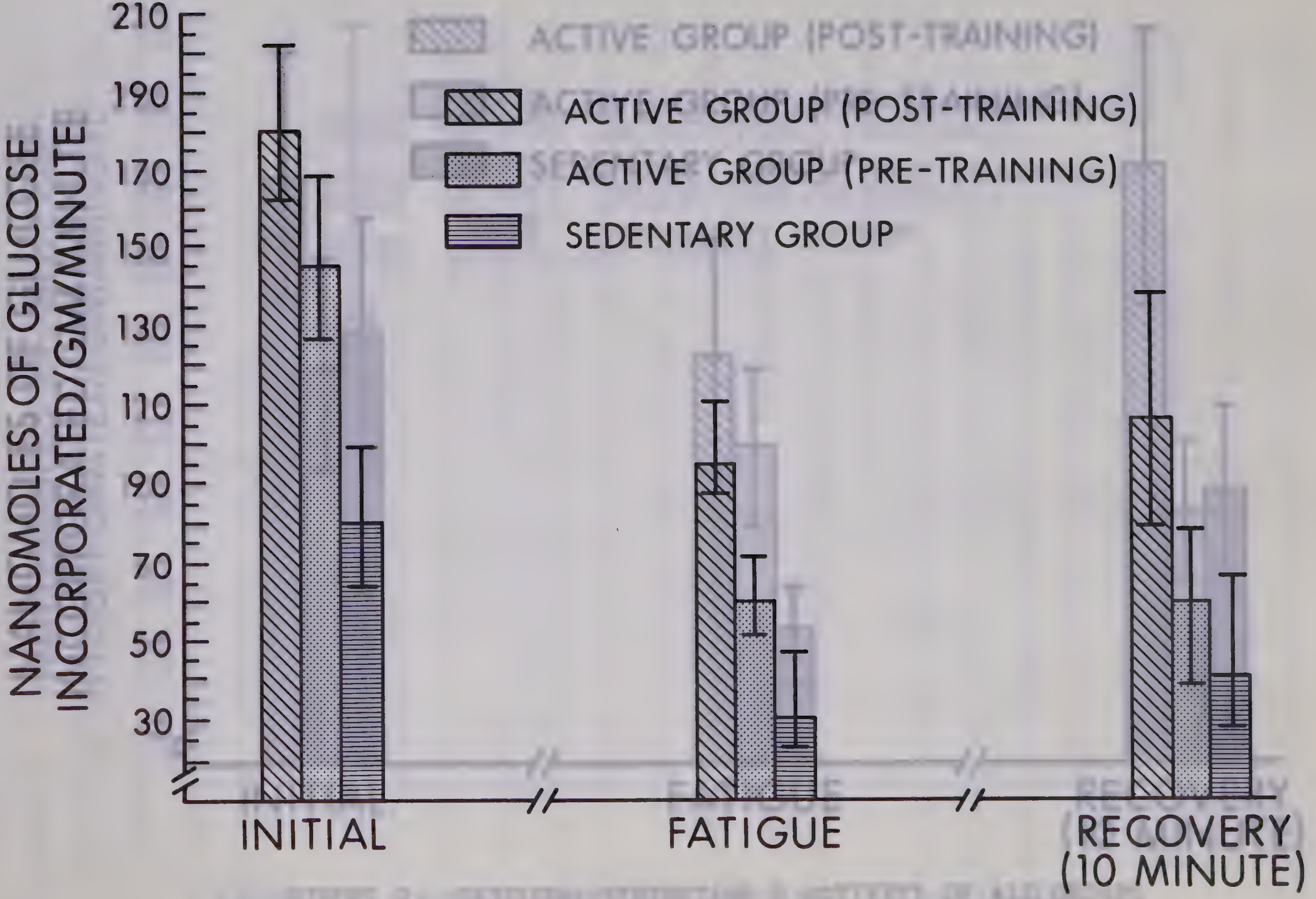


FIGURE 1. GLYCOGEN SYNTHETASE D ACTIVITY OF ALL GROUPS FOR SUBMAXIMAL WORK LOADS

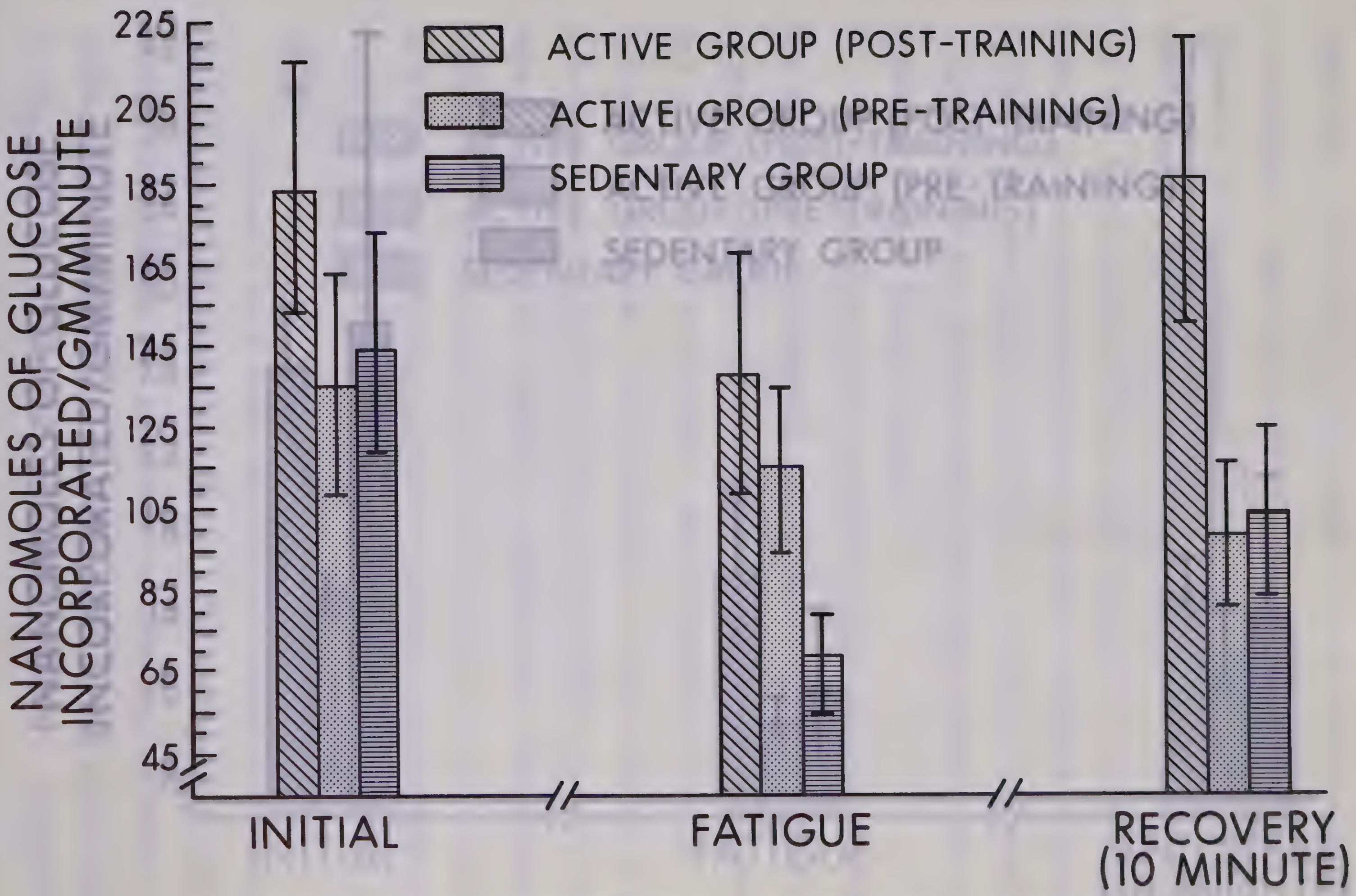


FIGURE 2. GLYCOGEN SYNTHETASE D ACTIVITY OF ALL GROUPS FOR MAXIMAL WORK LOADS

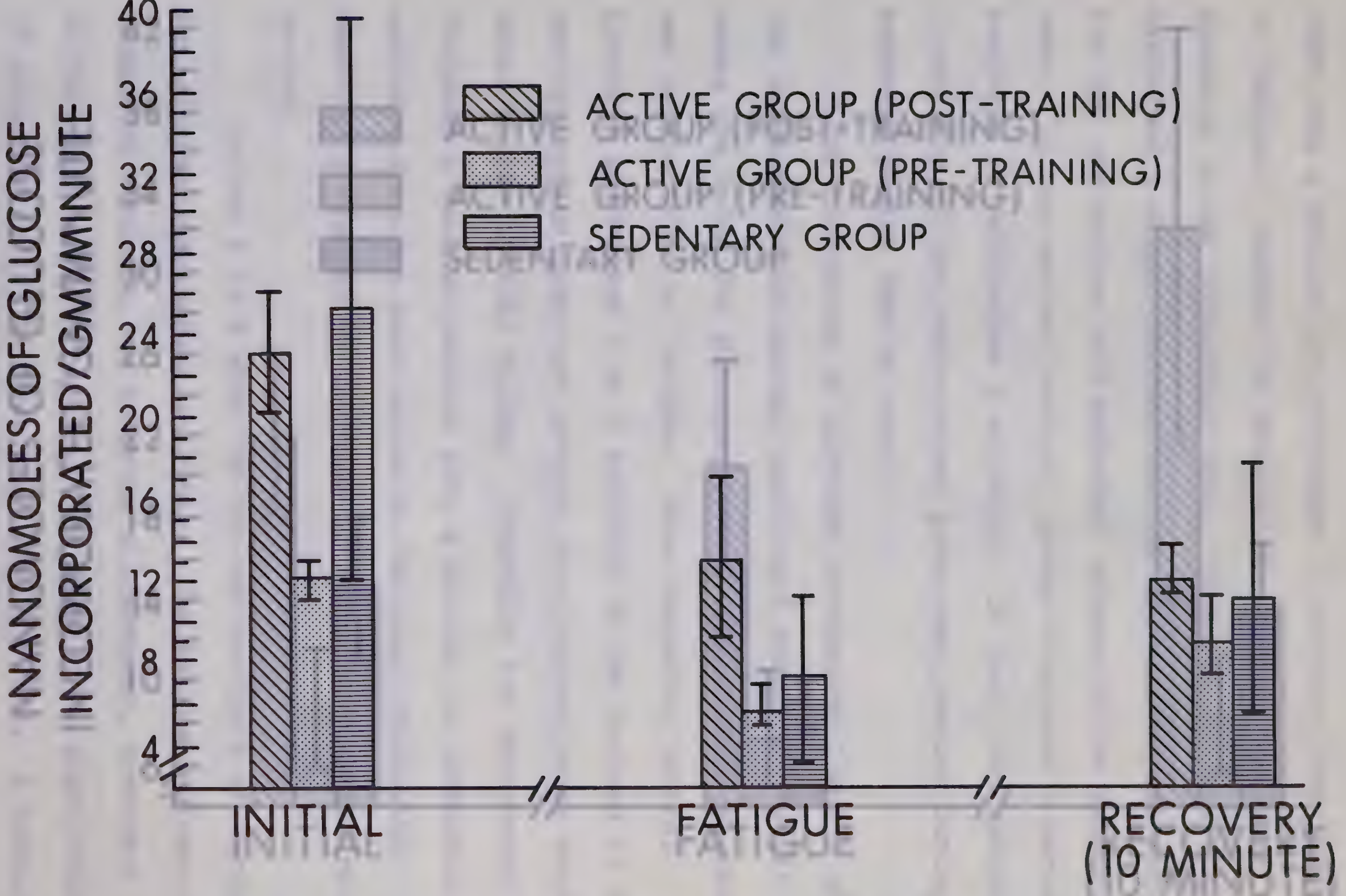


FIGURE 3. GLYCOGEN SYNTHETASE I ACTIVITY OF ALL GROUPS FOR SUBMAXIMAL WORK LOADS

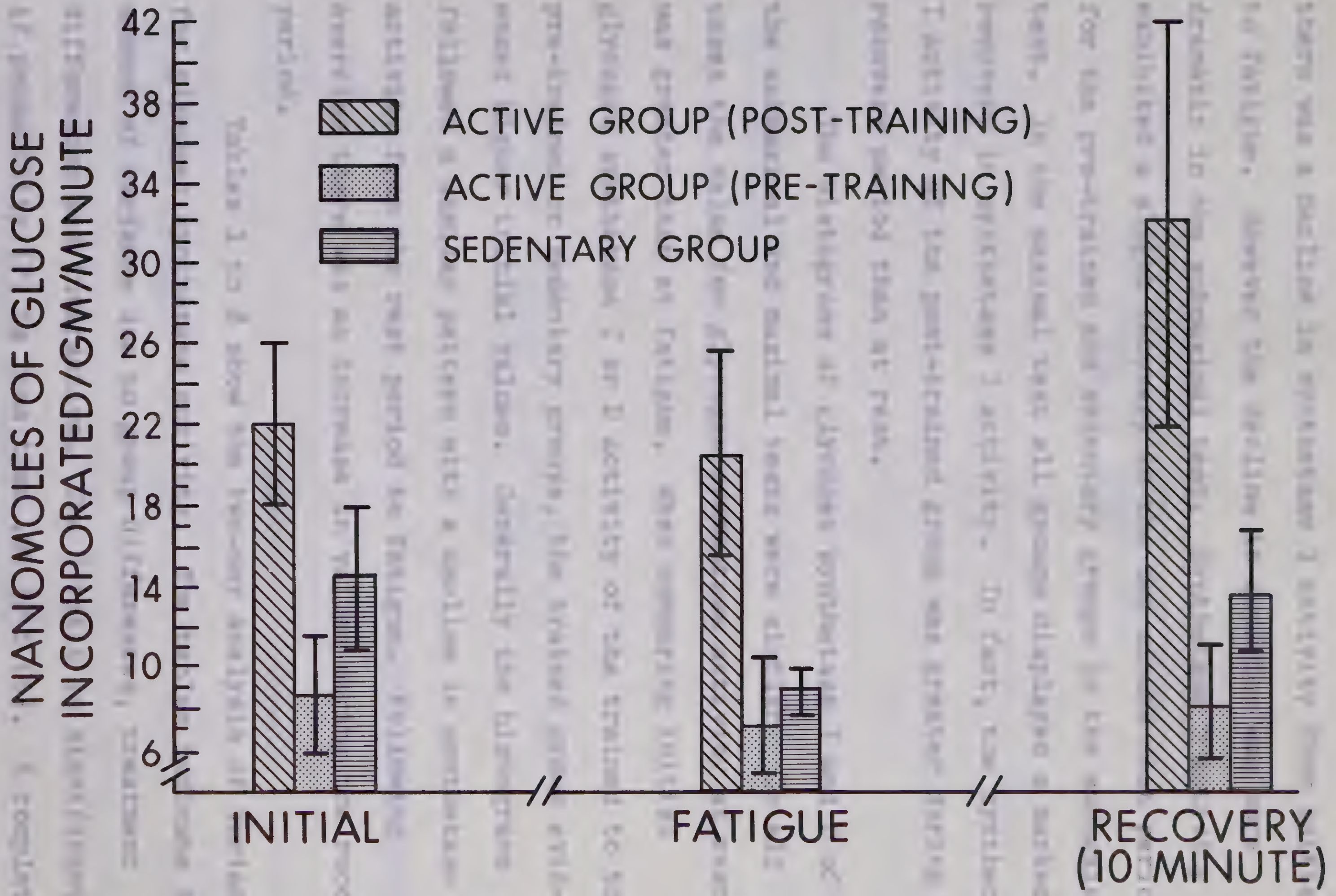


FIGURE 4. GLYCOGEN SYNTHETASE I ACTIVITY OF ALL GROUPS FOR MAXIMAL WORK LOADS

there was a decline in synthetase I activity from initial to fatigue. However the decline in activity was more dramatic in the submaximal test. Synthetase I activity exhibited a slight recovery in the ten minute rest period for the pre-trained and sedentary groups in the submaximal test. In the maximal test all groups displayed a marked recovery in synthetase I activity. In fact, the synthetase I activity of the post-trained group was greater during the recovery period than at rest.

The histograms of glycogen synthetase I and D of the submaximal and maximal tests were similar. In all cases the value for glycogen synthetase activity at rest was greater than at fatigue. When comparing initial glycogen synthetase I or D activity of the trained to the pre-trained or sedentary groups, the trained group evidenced higher initial values. Generally the histograms followed a similar pattern with a decline in synthetase activity from the rest period to fatigue. Following exercise there was an increase in values during the recovery period.

Tables 1 to 8 show the two-way analysis of variance for glycogen synthetase activity. The tables indicate the amount of variance due to group differences, treatment differences and interaction. The F ratio is significant if probability (P) is less than .05 ($P < .05$). A complete summary of the analysis of variance may be found in the Appendix.

TABLE 1 ANALYSIS OF VARIANCE FOR GLYCOGEN SYNTHETASE I: SUBMAXIMAL: PRE VS. POST

SOURCE	M.S.	F	P
Groups (Pre vs. Post)	470.90	8.7*	0.030
Treatments	222.90	9.9*	0.004
Interaction (Groups by Treatments)	42.49	1.9	0.200

TABLE 2 ANALYSIS OF VARIANCE FOR GLYCOGEN SYNTHETASE I: MAXIMAL: PRE VS. POST

SOURCE	M.S.	F	P
Groups (Pre vs. Post)	2,520.00	10.58*	0.02
Treatments	118.20	0.75	0.50
Interaction (Groups by Treatments)	108.40	0.80	0.48

* F ratio significant at the 0.05 level

TABLE 3 ANALYSIS OF VARIANCE FOR GLYCOGEN SYNTHETASE D: SUBMAXIMAL: PRE VS. POST

SOURCE	M.S.	F	P
Groups (Pre vs. Post)	21,744.3	9.74*	0.010
Treatments	38,305.8	12.19*	0.001
Interaction (Groups by Treatments)	602.5	0.27	0.760

TABLE 4 ANALYSIS OF VARIANCE FOR GLYCOGEN SYNTHETASE D: MAXIMAL PRE VS. POST

SOURCE	M.S.	F	P
Groups (Pre vs. Post)	38,160.0	2.57	0.14
Treatments	4,892.0	1.50	0.25
Interaction (Groups by Treatments)	4,717.0	4.71*	0.02

* F ratio significant at the 0.05 level

TABLE 5 ANALYSIS OF VARIANCE FOR GLYCOGEN SYNTHETASE I: SUBMAXIMAL: SED VS. POST

SOURCE	M.S.	F	P
Groups (Sed vs. Post)	17.4	0.03	0.88
Treatments	710.6	6.70*	0.01
Interaction (Groups by Treatments)	58.9	0.56	0.58

TABLE 6 ANALYSIS OF VARIANCE FOR GLYCOGEN SYNTHETASE I: MAXIMAL SED VS. POST

SOURCE	M.S.	F	P
Groups (Sed vs. Post)	1,368.9	7.6*	0.02
Treatments	197.6	1.3	0.31
Interaction (Groups by Treatments)	77.3	0.5	0.61

* F ratio significant at the 0.05 level

TABLE 7 ANALYSIS OF VARIANCE FOR GLYCOGEN SYNTHETASE D: SUBMAXIMAL: SED VS. POST

SOURCE	M.S.	F	P
Groups (Sed vs. Post)	75,908.9	10.90*	0.0040
Treatments	19,471.7	11.30*	0.0001
Interaction (Groups by Treatments)	1,678.0	0.98	0.3900

TABLE 8 ANALYSIS OF VARIANCE FOR GLYCOGEN SYNTHETASE D: MAXIMAL SED VS. POST

SOURCE	M.S.	F	P
Groups (Sed vs Post)	55,982.8	3.5	0.080
Treatments	17,709.2	8.1*	0.001
Interaction (Groups by Treatments)	2,293.6	1.1	0.360

* F ratio significant at the 0.05 level

In several cases the amount of variance due to group differences as well as experimental error was significant, indicating a difference between groups. The amount of variance due to treatment differences as well as experimental error was, in most cases, significant indicating a notable difference between treatment means. In all but one case, interaction was not significant.

Multiple comparisons, which are illustrated by Tables 9 to 16, were made using Tukey's W procedure. The comparisons were between treatments within a group and treatments between groups. When comparing treatment means within the post-training group in the submaximal test there was a significant difference between the initial glycogen synthetase D activity and the fatigue values. However, neither the sedentary nor pre-training group displayed this difference. There were no significant differences between treatment means within the sedentary, pre and post-training groups for glycogen synthetase I activity in the submaximal test. The treatment means for glycogen synthetase I or D activity within the sedentary, pre and post-training groups of the maximal test did not differ significantly. There was a significant difference for treatments between groups in only two cases. The initial glycogen synthetase D values of the sedentary group differed significantly with those of the post-training group in the submaximal test.

TABLE 9 Q VALUES:
GLYCOGEN SYNTHETASE D:
SUBMAXIMAL: SED VS. POST

	Sed			Post		
	I_1	F_1	R_1	I_2	F_2	R_2
I_1						
F_1	3.27[a]					
R_1	2.41	0.85				
I_2	7.04*					
F_2		4.55		5.75*		
R_2			4.69	4.76	0.99	

TABLE 10 Q VALUES:
GLYCOGEN SYNTHETASE D:
MAXIMAL: SED VS. POST

	Sed			Post		
	I_1	F_1	R_1	I_2	F_2	R_2
I_1						
F_1	5.03[a]					
R_1	2.65	2.39				
I_2	2.50					
F_2		4.67		2.87		
R_2			5.26	0.10	2.97	

a = Q Values

* = Q Values significant at the 0.01 level

TABLE 11 Q VALUES:
GLYCOGEN SYNTHETASE I:
SUBMAXIMAL: SED VS. POST

	Sed			Post		
	I_1	F_1	R_1	I_2	F_2	R_2
I_1						
F_1	4.4					
R_1	3.4	1.0				
I_2	0.67					
F_2		1.43		2.31		
R_2			0.24	2.50	0.19	

TABLE 12 Q VALUES:
GLYCOGEN SYNTHETASE I:
MAXIMAL: SED VS. POST

	Sed			Post		
	I_1	F_1	R_1	I_2	F_2	R_2
I_1						
F_1	1.02[a]					
R_1	0.08	0.94				
I_2	1.52					
F_2		2.21		0.33		
R_2			3.50	1.90	2.23	

a = Q Values

* = Q Values significant at the 0.01 level

TABLE 13 Q VALUES:
GLYCOGEN SYNTHETASE D:
SUBMAXIMAL: PRE VS. POST

	Pre			Post		
	I_1	F_1	R_1	I_2	F_2	R_2
I_1						
F_1	4.97[a]					
R_1	5.12	0.15				
I_2	1.95					
F_2		2.35		4.61		
R_2			3.39	3.81	0.79	

TABLE 14 Q VALUES:
GLYCOGEN SYNTHETASE D:
MAXIMAL: PRE VS. POST

	Pre			Post		
	I_1	F_1	R_1	I_2	F_2	R_2
I_1						
F_1	1.37[a]					
R_1	2.52	1.15				
I_2	1.90					
F_2		0.96		2.90		
R_2			3.52	0.11	3.00	

a = Q Values

* = Q Values significant at the 0.01 level

TABLE 15 Q VALUES: GLYCOGEN SYNTHETASE I: SUBMAXIMAL: PRE VS. POST

	Pre			Post		
	I_1	F_1	R_1	I_2	F_2	R_2
I_1						
F_1	3.3[a]					
R_1	1.6	1.7				
I_2	4.6					
F_2		3.2		5.0		
R_2			1.4	5.4	0.4	

TABLE 16 Q VALUES: GLYCOGEN SYNTHETASE I: MAXIMAL: PRE VS. POST

	Pre			Post		
	I_1	F_1	R_1	I_2	F_2	R_2
I_1						
F_1	0.23[a]					
R_1	0.09	0.14				
I_2	2.54					
F_2		2.44		0.34		
R_2			4.45	1.95	2.29	

a = Q Values

* = Q Values significant at the 0.01 level

Tables 17 and 18 represent tests on overall differences between initial, fatigue and recovery. In both sedentary versus post-training and pre-training versus post-training, there was an overall significant difference between initial and fatigue glycogen synthetase D activity for the submaximal test. The overall difference between initial and fatigue for glycogen synthetase I was significant at the .05 level. In the maximal test the difference between initial and fatigue for glycogen synthetase I and D was not significant. However the overall difference between fatigue versus recovery for synthetase D activity was significant.

TABLE 17 TESTS ON OVERALL DIFFERENCES: PRE VS. POST

	Initial-Fatigue	Initial-Recovery	Fatigue-Recovery
Synthetase D (submaximal)	6.25[a]*	5.83	0.42
Synthetase D (maximal)	2.44	1.38	1.06
Synthetase I (submaximal)	5.87**	4.95**	0.93
Synthetase I (maximal)	0.39	1.27	1.65

TABLE 18 TESTS ON OVERALL DIFFERENCES: SED VS. POST

	Initial-Fatigue	Initial-Recovery	Fatigue-Recovery
Synthetase D (submaximal)	6.37*	5.07	1.31
Synthetase D (maximal)	2.50	4.67**	5.26*
Synthetase I (submaximal)	4.74**	4.17	0.57
Synthetase I (maximal)	0.95	1.29	2.24

a = Q Values

* = Q Values significant at the 0.01 level

** = Q Values significant at the 0.05 level

CHAPTER V

DISCUSSION OF RESULTS

The glycogen content of skeletal muscle has been cited as increasing with training (27, 40, 53). Glycogen synthetase, being the enzyme responsible for synthesis of the glycogen chain, may increase in activity or amount as a result of training. Several investigators have shown that adaptation of skeletal muscle to exercise and training occurred through increased activities of enzyme systems (7, 32, 34).

Danforth (20) and other researchers (2, 25, 54) have established that glycogen synthetase exists in two forms (I and D). Glycogen controls its' own synthesis by a mechanism which affects the interconversion of glycogen synthetase (I⟷D) in such a manner as to favour glycogenesis when tissue concentrations are low and to retard synthesis when tissue concentrations are high (20). In the present study, muscle glycogen levels were relatively high prior to exercise. As expected, most of the synthetase was present in the D form.

No literature demonstrating the effects of a training program upon the glycogen synthetase activity of human subjects was available. The skeletal muscle of trained guinea pigs has been shown to increase in (I) and in (I + D) enzyme activity in both red and white muscle (33).

Similarly, Lamb et al (39) discovered that the total glycogen synthetase activity was greater in the hearts of trained animals forty-eight hours after running than for the sedentary group at rest.

In the submaximal test, the post-training group exhibited higher initial glycogen synthetase I and D values than either the pre-training or sedentary groups. Only the glycogen synthetase D activity was significantly higher. There was no significant difference between pre versus post-training for initial glycogen synthetase I and D activity.

During submaximal exercise a decrease in muscle glycogen has been reported (4, 40, 53, 55). In the course of a short tetanus, the rate of glycogenolysis may have increased several hundred times. These changes in activity were paralleled by a corresponding rise in the phosphorylase-a content of muscle. The increased phosphorylase activity may have resulted in decreasing glycogen synthetase values by lessening the stability of the enzyme due to the disappearance of substrate (glycogen) (19). Moreover, Leloir et al (43) have indicated that the best primer for glycogen synthetase activity is glycogen and synthetase activity decreases with glycogen degradation. In this study, both glycogen synthetase I and D values decreased from initial to fatigue for all groups. Thus with the breakdown of glycogen a decrease in enzymatic activity

occurred. A significant decrease in glycogen synthetase D activity for the post-training group also occurred. The comparison made between initial and fatigue within groups approached significance at the .01 level. This was substantiated by the tests on overall differences in which the decrease in glycogen synthetase D activity between initial and fatigue was significant.

Several studies have demonstrated that glycogen undergoes a supercompensation effect following exercise (13, 38, 55). Lamb (38) demonstrated that an acute bout of exercise seemed nearly as effective as chronic exercise in stimulating glycogen supercompensation. Bergstrom and Hultman (13) concluded that exercise with glycogen depletion enhanced the resynthesis of glycogen. The factor operated locally in the exercised muscle and lasted at least three days. Glycogen supercompensation may have been caused by some exercised induced factor which either activated or stimulated the synthetase activity (38). In the submaximal test, muscle samples were taken ten minutes after the cessation of exercise. There were no significant increases in glycogen synthetase I or D activity from fatigue to recovery although in several examples there was a slight increase in enzymatic activity following fatigue. Quite possibly the ten minute recovery period was not sufficient time to experience a dramatic rise in synthetase activity which would be expected to coincide with glycogen supercompensation.

Glycogen expenditure during a brief intensive effort has previously been shown to be minimal (40). In the present study, maximal exercise usually lasted five to ten minutes before fatigue developed. In most cases initial glycogen synthetase I and D activity was greater for the post-training group as compared with the sedentary and pre-training groups. At the point of fatigue, there was a slight decrease in both glycogen synthetase I and D activity from the initial values. Within the ten minute recovery period for the maximal test, synthetase I and D values increased above the fatigue levels. In fact, the trained group demonstrated an increase in both synthetase I and D activity above the resting levels. This finding would agree with the work of Canal and Frattola (17) that immediately following exercise a marked increase in both glycogen synthetase I and D activity occurs. Perhaps a more significant rise in enzymatic activity would have occurred with a longer recovery period. During the recovery period there was a much greater increase in synthetase I and D activity in the maximal test when compared with the submaximal test. Perhaps a brief, acute bout of exercise is more effective than prolonged exercise in stimulating an increase in synthetase activity above the resting values.

Staneloni and Piras (58) demonstrated that electrical stimulation of rat skeletal muscle _in situ_ resulted in an

I to D conversion which took place within the first five seconds of stimulation. During recovery, glycogen synthetase D was converted to the more active I form. During recovery, muscle samples of the human subject in this study exhibited a decrease in enzymatic activity. Once again the brief duration of the recovery period may not have been sufficient to observe the reported D to I conversion following fatigue.

This study is the first to compare glycogen synthetase activity of working human skeletal muscle in the same subjects under submaximal and maximal exercise conditions and to consider the influence of training on this activity.

CHAPTER VI

SUMMARY AND CONCLUSIONS

The purpose of this thesis was to investigate the glycogen synthetase activity of working human skeletal muscle and the adaptations of the muscle in a trained and non-trained state to submaximal and maximal work loads.

A group of eighteen male subjects from the University of Alberta were divided into sedentary and active groups. Prior to the actual experiment all subjects were tested for MVO_2 on a bicycle ergometer. The work load of 70% MVO_2 was then calculated for the submaximal test. The work load for the maximal test was set at 100% MVO_2. All subjects underwent both the submaximal and maximal tests, during which, muscle biopsies were taken from the vastus lateralis. In the submaximal test biopsies were taken at rest, after every twenty minutes of work, at the point of fatigue, and after a ten minute recovery period. For the maximal test biopsies were taken at rest, at the point of fatigue and following a ten minute recovery. Simultaneously as muscle biopsies were taken, blood was withdrawn from the brachial vein. The active group, comprising of wrestlers and track men, trained for four to five months in their respective sports and then all physiological measures were taken again. At the cessation of all testing, muscle samples were analyzed for glycogen synthetase activity and glycogen content.

Blood samples were analyzed for glucose and lactic acid.

The trained group exhibited a significant decrease in glycogen synthetase D activity with submaximal exercise. However there was a decrease in glycogen synthetase activity from initial to fatigue in all cases. No significant differences were noted for fatigue versus recovery. Initial glycogen synthetase D activity of the post-training group was significantly greater than initial values of the sedentary group. As well, initial glycogen synthetase activity of the post-training group was higher than initial values of the sedentary or pre-training groups.

For the maximal test no significant decreases in glycogen synthetase activity from initial to fatigue were observed. A marked difference was noted between the initial glycogen synthetase activity of the post-training group and the sedentary and pre-training groups. However, no significant differences were observed between the fatigue and recovery values despite an increase in glycogen synthetase I and D above the resting level. Glycogen synthetase I and D activities were lower for the submaximal work test than for the maximal work test at the point of fatigue.

Conclusions

Training stimulated an increase in glycogen synthetase I and D activity in human skeletal muscle. Exercise to fatigue resulted in a decrease in activity of both forms of the enzyme.

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APPENDIX A

NAME ______________ Birth Date __________ Ht. _____ Ph _________

MVO$_2$ Date __________ Time __________ Wt. __________

	Work Load	VO$_2$	STPD factor
1			
2			
3			
4			
5			
6			
7			

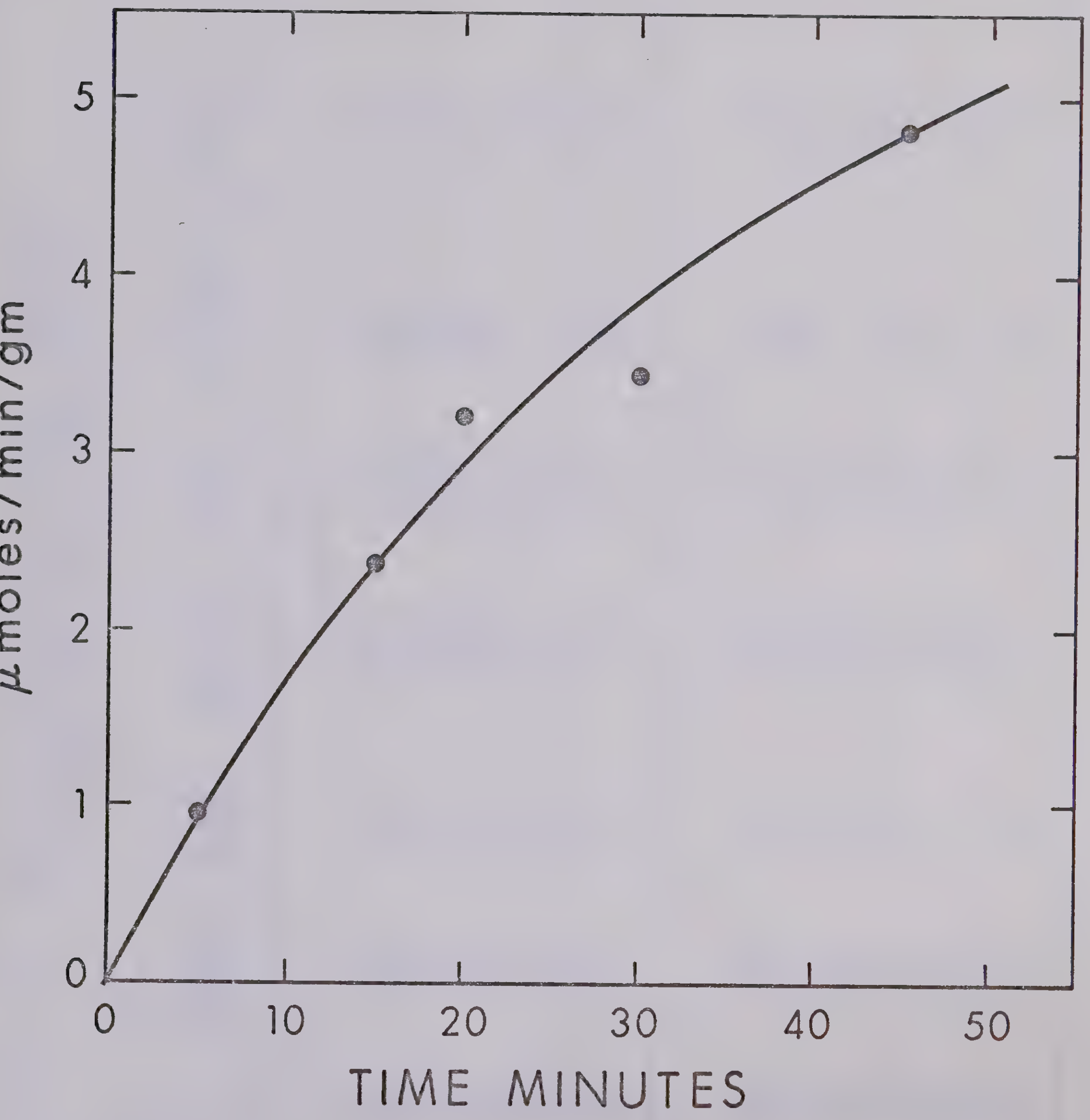
b Max Biopsy Date ______ Time __________ Wt. __________

ample	Period	Synth		Amylo		BL	BG	HR	VO$_2$	G	
		Wt.	%	Wt.	%					Wt.	%
1											
2											
3											
4											
5											
6											
7											
8											
9											

Biopsy Date ______ Time __________ Wt. __________

ample	Period	Synth.		Amylo		BL	BG	HR	VO$_2$	G	
		Wt.	%	Wt.	%					Wt.	%
1											
2											
3											
4											

1. DATA SHEET



2. TIME COURSE FOR SYNTHETASE D SOLUTION

3. TABLE OF AGE, WEIGHT, HEIGHT, SUBMAXIMAL AND MAXIMAL WORK LOADS, MVO_2 AND SUBMAXIMAL AND MAXIMAL WORK TIMES

Subj.	Age	Weight (lbs.)	Height (ins.)	Submax. Work Load	MVO_2 1/min.	Max. Work Load	Submax. Work Time (min.)	Max. Work Time (sec.)
				SEDENTARY				
B.H.	22	150	68	800	3.32	1320	60	370
B.K.	21	175	69	1100	3.65	1350	40	540
G.R.	19	160	69	900	3.25	1440	80	540
L.Do.	18	165	69	1050	3.63	1560	100	565
L.Du.	25	185	70	1100	3.60	1440	60	660
B.L.	20	170	71	1000	3.07		60	
E.B.	20	157	70	1100	3.50		100	
L.W.	19	179	71	1000	2.92	1560	80	600
J.K.	17	135	66	900	3.00	1320	80	450
				ACTIVE (Pre-training)				
D.C.	26	221	72	1050	2.80	1560	60	660
J.S.	22	165	74	1100	3.10	1560	80	720
D.I.	23	170	73	1100	3.20	1620	100	300
G.W.	28	142	66	1100	3.38		71	
L.B.	21	175	75	1200	3.05	1620	100	840
R.D.	24	160	68	1260	3.58	1800	100	480
S.G.	26	151	67	1300	3.40	1800	56	600
B.T.	25	168	69	1050	2.90		76	
G.V.	26	180	74	1100	3.45	1800	100	420
B.M.	22	153	69	1100	3.33	1620	80	840

..... continued

Table 3. continued ...

Subj.	Age	Weight (lbs.)	Height (ins.)	Submax. Work Load	MVO_2 1/min.	Max. Work Load	Submax. Work Time (min.)	Max. Work Time (sec.)
ACTIVE (Post-training)								
D.C.	26	210	72	1320	4.00	1680	60	880
J.S.	23	160	74	1320	4.10	1680	72	480
D.I.	23	172	73	1200	4.00	1620	72	280
G.W.	28	139	66	1200	4.18	1800	60	405
L.B.	21	177	75	1320	4.48	1680	93	720
R.D.	24	160	68	1260	4.30	1800	60	410
S.G.	26	158	67	1260	4.35	1800	60	535
B.T.	26	162	69	1260	4.42	1800	60	570

APPENDIX B

1. UNIVERSITY OF ALBERTA
DEPARTMENT OF MEDICINE AND
FACULTY OF PHYSICAL EDUCATION

CONSENT TO PARTICIPATE IN A RESEARCH STUDY

SUBJECT . . . . DATE . . . . . . .TIME A.M. P.M.

1. I agree to participate in an investigation and in relation to this hereby authorize Drs. . . . . and/or such assistants as may be selected by them, to perform the following procedure(s):

. . . .

. . . .

. . . .

2. Drs. . . . . have explained the purpose of this study and I understand this routine of the procedure outlined above.

. . . .
Witness Signature of Subject

If the subject is unable to sign or is under 21 years of age, complete the following:

The subject is a minor (. . . . . years of age).

or

The subject is unable to sign because . . . .

. . . .

As the closest relative or legal guardian I hereby sign on his/her behalf:

. . . .
Witness Signature Relationship

2. MUSCLE GLYCOGEN SYNTHETASE D ACTIVITY EXPRESSED AS NANOMOLES OF GLUCOSE-C^{14} INCORPORATED/GM/MIN. FOR SUBMAXIMAL TEST

Subject	Time at which sample was taken						
	0(Initial)	20	40	60	80	Fatigue	Recovery (10 minutes)
				SEDENTARY			
B.H.	75	147	3			36	9
B.K.	76	22				13	38
G.R.	33	25	22			4	11
L.Do.	56	169	148	30	79	12	38
L.Du.	102	85	84			57	34
B.L.	195	73	130			117	207
E.B.	80	78	35	18	17	29	32
L.W.	24	5	25	91		35	47
J.K.	78	48	41	124*		10	4
			ACTIVE (Pre-training)				
D.C.	73	85	168			102	59
J.S.	179	39	58	6	4	3	5
D.I.	120	163	107	48	189*	61	34
G.W.	109	97	14	154		40	30
L.B.	243	154	115	50		61	211
R.D.	229	103	155	156	123	44	57
S.G.	73	120	75			75	36
G.V.	98	141	147	96	47	92	38
B.M.	195	296	141	133	118	70	56

..... continued

Table 2. continued ...

Subject	Time at which sample was taken 0(Initial)	20	40	60	80	Fatigue	Recovery (10 minutes)
			ACTIVE (Post-training)				
D.C.	214	158	257			70	15
J.S.	177	49	52	181*		30	121
D.I.	213	359	147	162		88	17
G.W.	55	11	61	7		111	16
L.B.	240	103	111	7	217*	147	285
R.D.	135	74	62			83	77
S.G.	155	92	178	45		97	189
B.T.	228	196	583*			155	171

* Value is unacceptable

3. MUSCLE GLYCOGEN SYNTHETASE I ACTIVITY EXPRESSED AS NANOMOLES OF GLUCOSE-C^{14} INCORPORATED/GM/MINUTE FOR THE SUBMAXIMAL TEST

Subject	Time at which sample was taken						
	0(Initial)	20	40	60	80	Fatigue	Recovery (10 minutes)
				SEDENTARY			
B.H.	10	9	3			2	2
G.R.	15	11	9	3		1	5
L.Do.	16	13	18	14	12	5	3
L.Du.	12	6	8			2	6
L.W.	98	74	16	27		32	42
J.K.	4	1	2	9		2	11
			ACTIVE (Pre-training)				
D.C.	12	6	15			9	12
J.S.	14	18	10	11	5	7	7
D.I.	14	6	3	1	9	6	6
L.B.	7	1	6	3		5	6
R.D.	12	9	16	8	7	4	10
S.G.	14	13	3	3		4	14
			ACTIVE (Post-training)				
D.C.	18	5	21			7	10
J.S.	32	28	24	33		29	11
D.I.	24	14	12	27		10	12
L.B.	32	10	45	8	16	9	14
R.D.	12	12	10			3	11
G.W.	20	16	59	23		22	17

4. BLOOD GLUCOSE IN MG. % FOR THE SUBMAXIMAL TEST

Subject	Time at which sample was taken 0(Initial)	20	40	60	80	Fatigue	Recovery (10 minutes)
				SEDENTARY			
B.H.	86	63	86			58	65
B.K.	100	83				79	104
G.R.	91	90	58	52		56	63
L.Do.	98	73	87	91	70	75	88
L.Du.	111	115	91			85	81
B.L.	97	104	96			100	98
E.B.	77	83	77	75	65	94	94
L.W.	71	97	88	79		101	110
J.K.	72	98	100	112		51	88
			ACTIVE (Pre-training)				
D.C.	81	63	63			83	84
J.S.	80	77	84	73	69	80	84
D.I.	105	105	112	112	110	113	116
G.W.	61	61	77	73		60	63
L.B.	85	78	80	105		82	98
R.D.	93	77	88	95	90	84	98
S.G.	84	63	116*			78	84
B.T.	125	104	97	93		87	92
G.V.	51	83	70	88	90	84	102
B.M.	75	104	57	98	84	85	110

..... continued

Table 4. continued ...

Subject	Time at which sample was taken						
	0(Initial)	20	40	60	80	Fatigue	Recovery (10 minutes)
			ACTIVE (Post-training)				
D.C.	81	56	81			60	76
J.S.	72	67	63			55	67
D.I.	81	71	111*	92		85	69
G.W.	70	64	57			42	64
L.B.	90	84	86	108	127*	72	84
R.D.	74	88	81			100	79
S.G.	92	92	98			88	93
B.T.	87	98	100			72	76

* Value is unacceptable

5. BLOOD LACTATE IN MG. % FOR THE SUBMAXIMAL TEST

Subject	Time at which sample was taken						
	0(Initial)	20	40	60	80	Fatigue	Recovery (10 minutes)
				SEDENTARY			
B.H.	6	96	70			42	31
B.K.	12	69				113	69
G.R.	10	77	65	50		45	38
L.Do.	8	70	69	43	55	64	29
L.Du.	15	60	49			45	29
B.L.	5	56	64			50	30
E.B.	15	23	16	19	19	31	26
L.W.	14	64	64	43		36	26
J.K.	11	64	56	35		42	42
				ACTIVE (Pre-training)			
D.C.	9	51	51			54	32
J.S.	7	14	14	28		21	24
D.I.	14	57	36	52	22	31	18
G.W.	10	53	42	53		38	32
L.B.	6	81	72	79		30	38
R.D.	8	30	33	39	46	30	13
S.G.	10	78	55			78	34
B.T.	12	43	31	30		26	39
G.V.	12	54	49	28	61	72	28
B.M.	16	65	69	56	39	31	21

..... continued

Table 5. continued ...

Subject	Time at which sample was taken						
	0(Initial)	20	40	60	80	Fatigue	Recovery (10 minutes)
			ACTIVE (Post-training)				
D.C.	11	78	80			68	12
J.S.	7	30	22	24		44	20
D.I.	9	60	102	48		16	36
G.W.	14	130	114			54	36
L.B.	9	42	48	74	32	28	20
R.D.	13	78	78			76	38
S.G.	6	96	72			58	18
B.T.	9	58	52			50	36

6. MUSCLE GLYCOGEN SYNTHETASE D ACTIVITY EXPRESSED IN NANOMOLES OF GLUCOSE C^{14} INCORPORATED/GM/MIN. FOR THE MAXIMAL TEST

Subject	Initial	Fatigue	Recovery (10 min.)
SEDENTARY			
B.H.	107	53	181
B.K.	44	17	33
G.R.	66	61	48
L.Do.	172	119	191
L.Du.	102	112	142
L.W.	208	18	20
J.K.	316	87	111
ACTIVE (Pre-training)			
D.C.	85	70	58
J.S.	42	35	17
D.I.	135	74	55
L.B.	323	125	99
R.D.	80	99	109
S.G.	77	130	166
G.V.	175	135	173
B.M.	174	254	103
ACTIVE (Post-training)			
D.C.	85	56	52
J.S.	162	86	131
D.I.	344	368	444
G.W.	75	86	110
L.B.	322	159	172
R.D.	136	96	170
S.G.	178	149	225
B.T.	169	114	180

7. MUSCLE GLYCOGEN SYNTHETASE I ACTIVITY EXPRESSED IN NANOMOLES OF GLUCOSE C^{14} INCORPORATED/GM/MIN. FOR THE MAXIMAL TEST

Subject	Initial	Fatigue	Recovery(10 min.)
	SEDENTARY		
B.H.	8	7	9
G.R.	9	7	7
L.Do.	8	6	12
L.Du.	14	12	9
L.W.	31	12	20
J.K.	17	12	28
	ACTIVE (Pre-training)		
D.C.	6	2	2
J.S.	8	7	10
D.I.	22	26	23
L.B.	6	4	9
R.D.	2	2	2
S.G.	9	5	4
	ACTIVE (Post-training)		
D.C.	21	29	26
J.S.	35	34	17
D.I.	17	12	29
L.B.	12	10	77
R.D.	13	5	11
S.G.	36	34	32

8. BLOOD GLUCOSE IN MG% FOR THE MAXIMAL TEST

Subject	Initial	Fatigue	Recovery (10 min.)
		SEDENTARY	
B.H.	77	47	86
B.K.	79	83	122
G.R.	57	81	88
L.Do.	89	93	96
L.Du.	91	85	95
L.W.	71	76	81
J.K.	64	117	97
		ACTIVE (Pre-training)	
D.C.	103	99	115
J.S.	87	88	72
D.I.	91	101	99
L.B.	89	103	97
R.D.	89	99	117
S.G.	90	90	73
G.V.	71	88	85
B.M.	93	122	119
		ACTIVE (Post-training)	
D.C.	65	79	65
J.S.	76	73	76
D.I.	80	74	90
G.W.	91	81	66
L.B.	90	81	79
R.D.	76	70	77
S.G.	98	98	95
B.T.	74	92	83

9. BLOOD LACTATE IN MG% FOR THE MAXIMAL TEST

Subject	Initial	Fatigue	Recovery(10 min.)
SEDENTARY			
B.H.	12	104	118
B.K.	13	107	129
G.R.	6	112	78
L.Do.	25	119	81
L.Du.	16	81	29
L.W.	14	121	122
J.K.	11	148	124
ACTIVE (Pre-training)			
D.C.	8	70	63
J.S.	4	86	38
D.I.	10	62	47
L.B.	5	77	83
R.D.	9	76	63
S.G.	4	72	57
B.T.	4	72	57
G.V.	11	67	83
B.M.	7	114	71
ACTIVE (Post-training)			
D.C.	11	58	56
J.S.	7	78	56
D.I.	14	90	28
G.W.	9	102	42
L.B.	16	74	26
R.D.	11	110	114
S.G.	12	78	58
B.T.	10	94	84

APPENDIX C

1. SUMMARY OF ANALYSIS OF VARIANCE: SEDENTARY VS. POST-TRAINING GROUPS SUBMAXIMAL TEST - GLYCOGEN SYNTHETASE D

Source	SS	DF	MS	F	P
Between Subjects	187451.50	17			
'A' Main Effects (Groups)	75908.94	1	75908.94	10.89	0.005
Subjects within Groups	111542.44	16	6971.40		
Within Subjects	97228.88	36			
'B' Main Effects (Treatments)	38943.39	2	19471.69	11.34	0.0001
'A*B' Interaction	3356.09	2	1678.04	0.98	0.39
'B' X Subjects within Groups	54930.13	32	1716.57		

2. SUMMARY OF ANALYSIS OF VARIANCE: SEDENTARY VS. POST-TRAINING GROUPS MAXIMAL TEST - GLYCOGEN SYNTHETASE D

Source	SS	DF	MS	F	P
Between Subjects	313052.19	17			
'A' Main Effects (Groups)	55982.81	1	55982.81	3.48	0.08
Subjects within Groups	257069.00	16	16066.81		
Within Subjects	109720.00	36			
'B' Main Effects (Treatments)	35418.38	2	17709.19	8.13	0.001
'A*B' Interaction	4587.19	2	2293.59	1.05	0.36
'B' X Subjects within Groups	69715.00	32	2178.59		

3. SUMMARY OF ANALYSIS OF VARIANCE: SEDENTARY VS. POST-TRAINING GROUPS SUBMAXIMAL TEST - GLYCOGEN SYNTHETASE I

Source	SS	DF	MS	F	P
Between Subjects	6988.75	11			
'A' Main Effects (Groups)	17.36	1	17.36	0.03	
Subjects within Groups	6971.40	10	697.14		
Within Subjects	3662.00	24			
'B' Main Effects (Treatments)	1421.17	2	710.58	6.69	0.005
'A*B' Interaction	117.73	2	58.86	0.56	0.58
'B' X Subjects within Groups	2123.11	20	106.16		

4. SUMMARY OF ANALYSIS OF VARIANCE: SEDENTARY VS. POST-TRAINING GROUPS MAXIMAL TEST - GLYCOGEN SYNTHETASE I

Source	SS	DF	MS	F	P
Between Subjects	3155.33	11			
'A' Main Effects (Groups)	1368.99	1	1368.99	7.62	0.02
Subjects within Groups	1797.33	10	179.73		
Within Subjects	3690.67	24			
'B' Main Effects (Treatments)	395.16	2	197.58	1.26	0.31
'A*B' Interaction	154.52	2	77.25	0.49	0.62
'B' X Subjects within Groups	3141.01	20	157.05		

5. SUMMARY OF ANALYSIS OF VARIANCE: PRE-TRAINING VS. POST-TRAINING GROUPS SUBMAXIMAL TEST - GLYCOGEN SYNTHETASE D

Source	SS	DF	MS	F	P
Bet Subj	73246.81	8			
Subj W Group	73246.81	8	9155.85		
Within Subj	202795.38	45			
B	21744.31	1	21744.31	9.74	0.01
B X Subj W G	17862.38	8	2232.80		
C	76611.56	2	38305.78	12.19	0.0006
C X Subj W G	50283.63	16	3142.73		
BC	1205.06	2	602.53	0.27	0.76
BC X Subj W G	35088.69	16	2193.04		

6. SUMMARY OF ANALYSIS OF VARIANCE: PRE-TRAINING VS. POST-TRAINING GROUPS MAXIMAL TEST - GLYCOGEN SYNTHETASE I

Source	SS	DF	MS	F	P
Bet Subj	152561.00	8			
Subj W Group	152561.00	8	19070.13		
Within Subj	244240.00	45			
B	38160.00	1	38160.00	2.57	0.15
B X Subj W G	118603.00	8	14825.38		
C	9784.00	2	4892.00	1.50	0.25
C X Subj W G	52239.00	16	3264.94		
BC	9434.00	2	4717.00	4.71	0.025
BC X Subj W G	16020.00	16	1001.25		

7. SUMMARY OF ANALYSIS OF VARIANCE: PRE-TRAINING VS. POST-TRAINING GROUPS SUBMAXIMAL TEST - GLYCOGEN SYNTHETASE I

Source	SS	DF	MS	F	P
Bet Subj	244.61	5			
Subj W G	244.61	5	48.92		
Within Subj	1718.10	30			
B	470.89	1	470.89	8.72	0.03
B X Subj W G	270.11	5	54.02		
C	445.73	2	222.87	9.97	0.004
C X Subj W G	223.54	10	22.35		
BC	84.78	2	42.39	1.90	0.20
BC X Subj W G	223.04	10	22.30		

8. SUMMARY OF ANALYSIS OF VARIANCE: PRE-TRAINING VS. POST-TRAINING GROUPS MAXIMAL TEST - GLYCOGEN SYNTHETASE I

Source	SS	DF	MS	F	P
Bet Subj	1016.47	5			
Subj W Group	1016.47	5	203.29		
Within Subj	7098.94	30			
B	2520.04	1	2520.04	10.58	0.02
B X Subj W G	1190.73	5	238.15		
C	236.48	2	118.24	0.75	0.49
C X Subj W G	1579.53	10	157.95		
BC	216.74	2	108.37	0.80	0.48
BC X Subj W G	1355.40	10	135.54		

9. MULTIPLE COMPARISONS: GLYCOGEN SYNTHETASE D: SUBMAXIMAL SED VS. POST

A. Comparisons made are: I_1-F_1 I_1-R_1 F_1-R_1

MS for error is B x S(G) = 1716.6

Standard error is 13.8

Critical Q has K = 6 with DF = 32

Mean differences are: 45.1 33.3 11.8

Q values are: 3.2 2.4 0.9

B. Comparisons made are: I_2-F_2 I_2-R_2 F_2-R_2

MS for error is B x S(G) = 1716.6

Standard error is 13.8

Critical Q has K = 6 with DF = 32

Mean differences are: 79.4 65.7 13.7

Q values are: 5.7 4.8 1.0

C. Comparisons made are: I_1-I_2 F_1-F_2 R_1-R_2

MS for error is MS within (ie. pooled errors S(G) and B x S(G)) = 10.2

Standard error is 13.8

Critical Q has K = 6 with DF = 32

Mean differences are: 97.2 62.9 64.8

Q values are: 7.0 4.6 4.7

10. MULTIPLE COMPARISONS: GLYCOGEN SYNTHETASE D: MAXIMAL SED VS. POST

A. Comparisons made are: I_1-F_1 I_1-R_1 F_1-R_1

MS for error is B x S(G) = 2178.6

Standard error is 15.6

Critical Q has K = 6 with DF = 32

Mean differences are: 78.3 41.3 37.0

Q values are: 5.0 2.7 2.4

B. Comparisons made are: I_2-F_2 I_2-R_2 F_2-R_2

MS for error is B x S(G) = 2178.6

Standard error is 15.6

Critical Q has K = 6 with DF = 32

Mean differences are: 44.6 1.6 46.2

Q values are: 2.9 0.1 3.0

C. Comparisons made are: I_1-I_2 F_1-F_2 R_1-R_2

MS for error is MS within (ie. pooled errors S(G) and B x S(G)) = 22.3

Standard error is 15.6

Critical Q has K = 6 with DF = 32

Mean differences are: 38.9 72.6 81.8

Q values are: 2.5 4.7 5.3

11. MULTIPLE COMPARISONS: GLYCOGEN SYNTHETASE I: SUBMAXIMAL SED VS. POST

A. Comparisons made are: I_1-F_1 I_1-R_1 F_1-R_1

MS for error is B x S(G) = 106.2

Standard error is 4.2

Critical Q has K = 6 with DF = 20

Mean differences are: 18.5 14.3 4.2

Q values are: 4.4 3.4 1.0

B. Comparisons made are: I_2-F_2 I_2-R_2 F_2-R_2

MS for error is B x S(G) = 106.2

Standard error is 4.2

Critical Q has K = 6 with DF = 20

Mean differences are: 9.7 10.5 0.8

Q values are: 2.3 2.5 0.2

C. Comparisons made are: I_1-I_2 F_1-F_2 R_1-R_2

MS for error is MS within (ie. pooled errors S(G) and B x S(G)) = 2.5

Standard error is 4.2

Critical Q has K = 6 with DF = 20

Mean differences are: 2.8 6.0 1.0

Q values are: 0.7 1.4 0.2

12. MULTIPLE COMPARISONS: GLYCOGEN SYNTHETASE I: MAXIMAL SED VS. POST

A. Comparisons made are: I_1-F_1 I_1-R_1 F_1-R_1

MS for error is B x S(G) = 157.1

Standard error is 5.1

Critical Q has K = 6 with DF = 20

Mean differences are: 5.2 0.4 4.8

Q values are: 1.0 0.1 0.9

B. Comparisons made are: I_2-F_2 I_2-R_2 F_2-R_2

MS for error is B x S(G) = 157.1

Standard error is 5.1

Critical Q has K = 6 with DF = 20

Mean differences are: 1.7 9.7 11.4

Q values are: 0.3 1.9 2.2

C. Comparisons made are: I_1-I_2 F_1-F_2 R_1-R_2

MS for error is MS within (ie. pooled errors S(G) and B x S(G)) = 0.7

Standard error is 5.1

Critical Q has K = 6 with DF = 20

Mean differences are: 7.8 11.3 17.9

Q values are: 1.5 2.2 3.5

13. MULTIPLE COMPARISONS: GLYCOGEN SYNTHETASE D: SUBMAXIMAL PRE VS. POST

A. Comparisons made are: I1-F1 I1-R1 F1-R1
I2-F2 I2-R2 F2-R2

Pooled error mean square is 2667.9

Pooled error DF (normal) = 32

Standard error is 17.2

Critical Q has K = 6

Mean differences are: 85.7 88.2 2.5
79.4 65.8 13.7

Q values are: 5.0 5.1 0.1
4.6 3.8 0.8

B. Comparisons made are: I1-I2 F1-F2 R1-R2

Pooled error mean square is 2206.3

Pooled error DF (normal) = 24

Standard error is 15.7

Critical Q has K = 6

Mean differences are: 30.6 36.8 53.0

Q values are: 2.0 2.4 3.4

14. MULTIPLE COMPARISONS: GLYCOGEN SYNTHETASE D: MAXIMAL PRE VS. POST

A. Comparisons made are: I1-F1 I1-R1 F1-R1

I2-F2 I2-R2 F2-R2

Pooled error mean square is 2133.0

Pooled error DF (normal) = 32

Standard error is 15.4

Critical Q has K = 6

Mean differences are: 21.1 38.9 17.8

44.6 1.6 46.2

Q values are: 1.4 2.5 1.2

2.9 0.1 3.0

B. Comparisons made are: I1-I2 F1-F2 R1-R2

Pooled error mean square is 5609.3

Pooled error DF (normal) = 24

Standard error is 25.0

Critical Q has K = 6

Mean differences are: 47.5 24.0 88.0

Q values are: 1.9 1.0 3.5

15. MULTIPLE COMPARISONS: GLYCOGEN SYNTHETASE I: SUBMAXIMAL PRE VS. POST

A. Comparisons made are: I1-F1 I1-R1 F1-R1

I2-F2 I2-R2 F2-R2

Pooled error mean square is 22.3

Pooled error DF (normal) = 20

Standard error is 1.9

Critical Q has K = 6

Mean differences are: 6.4 3.0 3.4

9.7 10.5 0.8

Q values are: 3.3 1.6 1.7

5.0 5.4 0.4

B. Comparisons made are: I1-I2 F1-F2 R1-R2

Pooled error mean square is 32.9

Pooled error DF (normal) = 15

Standard error is 2.3

Critical Q has K = 6

Mean differences are: 10.8 7.5 3.3

Q values are: 5.9 4.9 0.9

16. MULTIPLE COMPARISONS: GLYCOGEN SYNTHETASE I: MAXIMAL PRE VS. POST

A. Comparisons made are: I1-F1 I1-R1 F1-R1

I2-F2 I2-R2 F2-R2

Pooled error mean square is 146.7

Pooled error DF (normal) = 20

Standard error is 4.9

Critical Q has K = 6

Mean differences are: 1.1 0.5 0.7

1.7 9.7 11.3

Q values are: 0.2 0.1 0.1

0.3 2.0 2.3

B. Comparisons made are: I1-I2 F1-F2 R1-R2

Pooled error mean source is 169.7

Pooled error DF (normal) = 15

Standard error is 5.3

Critical Q has K = 6

Mean differences are: 13.5 13.0 23.7

Q values are: 2.5 2.4 4.4

17. TEST ON OVERALL DIFFERENCES: I-F I-R F-R: SED VS. POST SUBMAXIMAL TEST - GLYCOGEN SYNTHETASE D

MS for error is MS for B x S(G) = 1716.6 with DF = 32

Standard error is 918

Overall means for I, F and R are: 128.5 66.3 79.0

Mean differences are: 62.3 49.5 12.8

Q values are: 6.4 5.1 1.3

18. TEST ON OVERALL DIFFERENCES: I-F I-R F-R: SED VS. POST MAXIMAL TEST - GLYCOGEN SYNTHETASE D

MS for error is MS for B x S(G) = 2178.6 with DF = 32

Standard error is 11.0

Overall means for I, F and R are: 164.5 103.0 144.6

Mean differences are: 61.5 19.9 41.6

Q values are: 5.6 1.8 3.8

19. TEST ON OVERALL DIFFERENCES: I-F I-R F-R: SED VS. POST SUBMAXIMAL TEST - GLYCOGEN SYNTHETASE I

MS for error is MS for B x S(G) = 106.2 with DF = 20

Standard error is 3.0

Overall means for I, F and R are: 24.4 10.3 12.0

Mean differences are: 14.1 12.4 1.7

Q values are: 4.7 4.2 0.6

20. TEST ON OVERALL DIFFERENCES: I-F I-R F-R: SED VS. POST MAXIMAL TEST - GLYCOGEN SYNTHETASE I

MS for error is MS for B x S(G) = 157.1 with DF = 20

Standard error is 3.6

Overall means for I, F and R are: 18.4 15.0 23.1

Mean differences are: 3.5 4.7 8.1

Q values are: 1.0 1.3 2.2

21. TESTS ON OVERALL DIFFERENCES: I-F I-R F-R: PRE VS. POST SUBMAXIMAL - GLYCOGEN SYNTHETASE D

MS for error is MS for C x S(G) = 3142.7 with DF = 16

Standard error is 13.2

Overall means for I, F and R are: 161.8 79.3 84.9

Mean differences are: 82.5 77.0 5.6

Q values are: 6.2 5.8 0.4

22. TESTS ON OVERALL DIFFERENCES: I-F I-R F-R: PRE VS. POST MAXIMAL - GLYCOGEN SYNTHETASE D

MS for error is MS for C x S(G) = 3264.9 with DF = 16

Standard error is 13.5

Overall means for I, F and R are: 160.1 127.3 141.5

Mean differences are: 32.9 18.6 14.2

Q values are: 2.4 1.4 1.1

23. TESTS ON OVERALL DIFFERENCES: I-F I-R F-R: PRE VS. POST SUBMAXIMAL - GLYCOGEN SYNTHETASE I

MS for error is MS for C x S(G) = 22.4 with DF = 10

Standard error is 1.4

Overall means for I, F and R are: 17.6 9.6 10.8

Mean differences are: 8.0 6.8 1.3

Q values are: 5.9 4.9 0.9

24. TESTS ON OVERALL DIFFERENCES: I-F I-R F-R: PRE VS. POST MAXIMAL - GLYCOGEN SYNTHETASE I

MS for error is MS for C x S(G) = 158.0 with DF = 10

Standard error is 3.6

Overall means for I, F and R are: 15.6 14.2 20.2

Mean differences are: 1.4 4.6 6.0

Q values are: 0.4 1.3 1.7

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